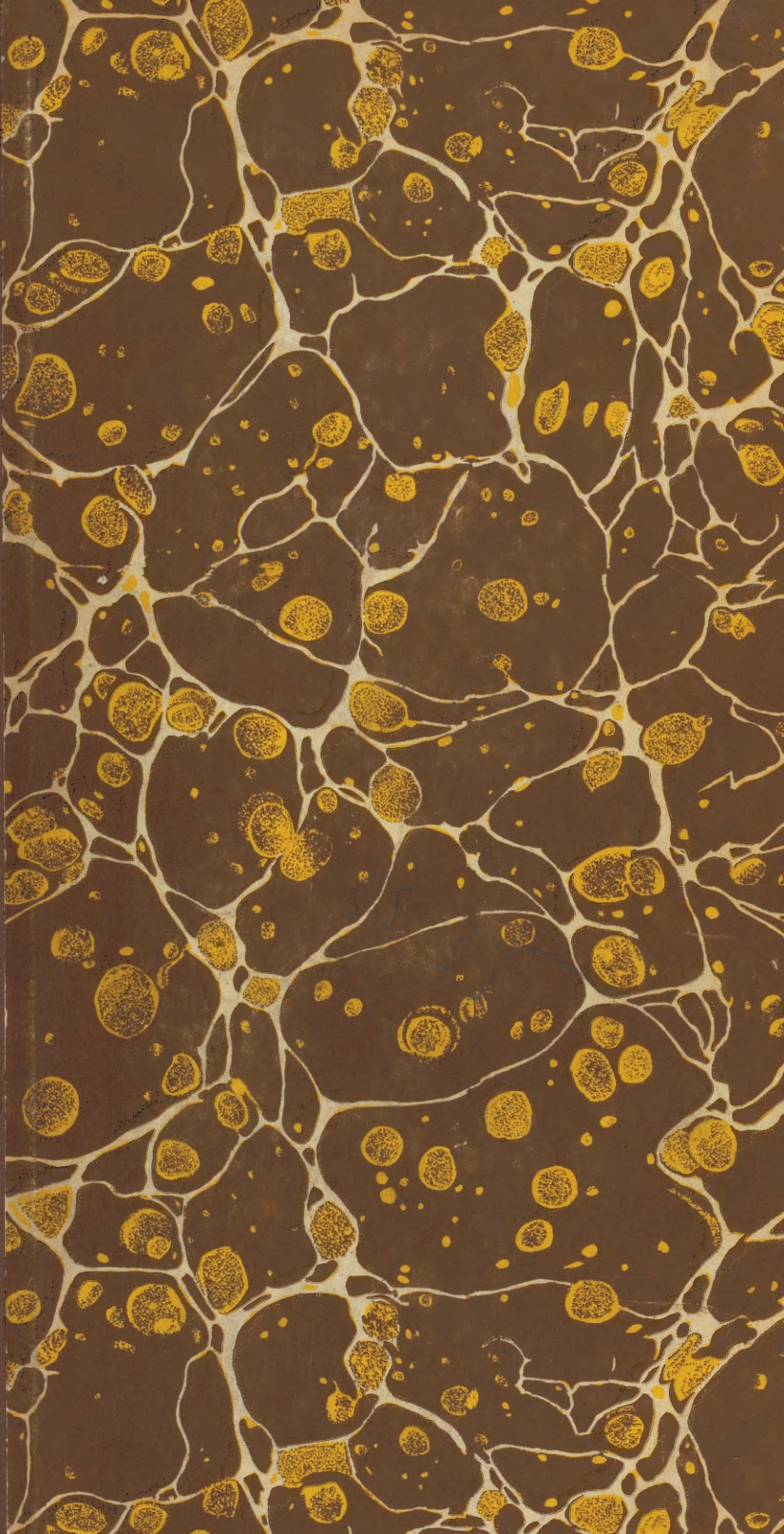




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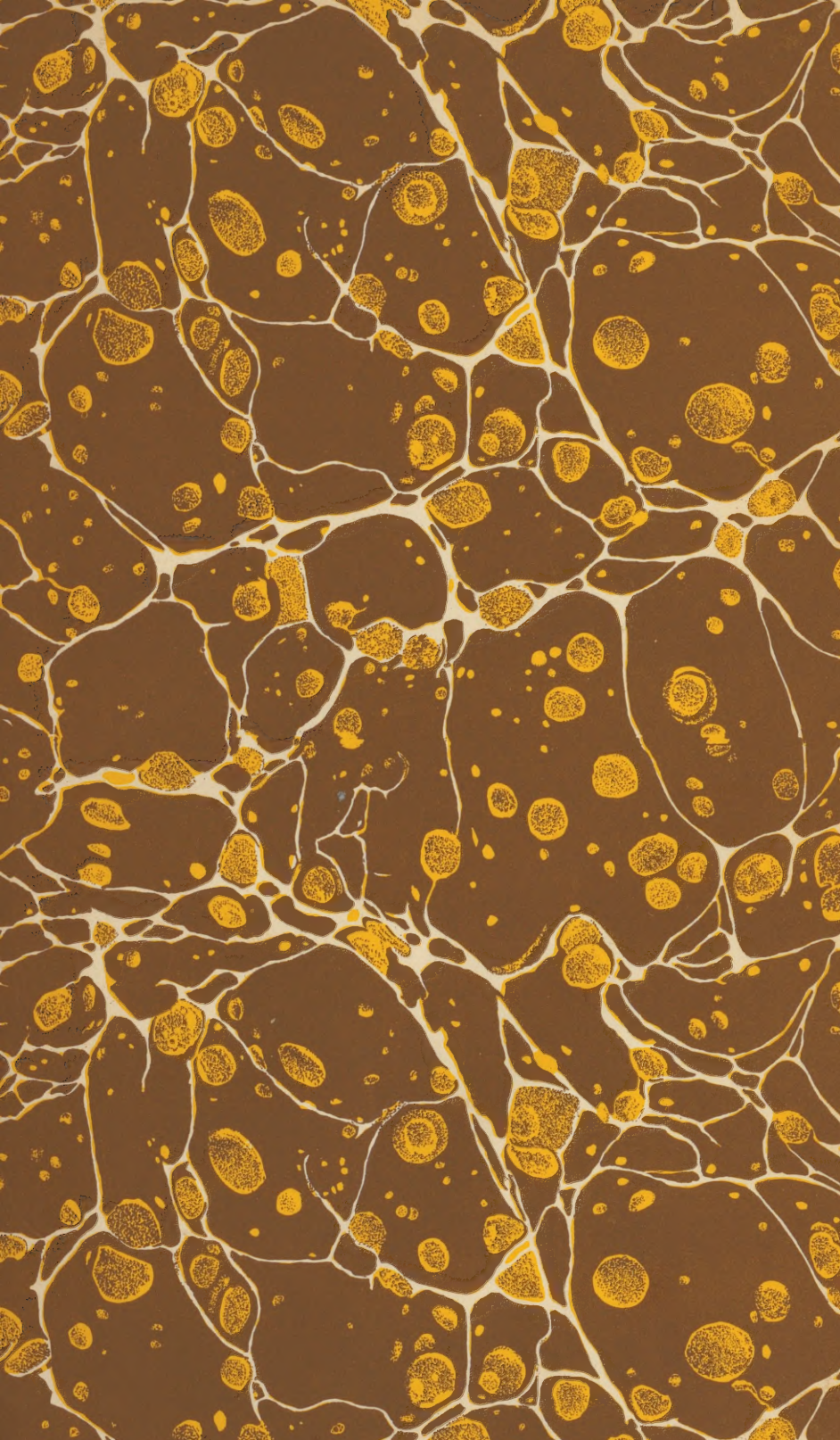
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A HAND-BOOK

OF

POST-MORTEM EXAMINATIONS

AND OF

MORBID ANATOMY.

BY

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PREFACE.

THIS work is intended to serve as a guide for those persons who may be called upon to perform post-mortem examinations. For most physicians this call is only an occasional one, so that they may feel the need of some hand-book to which they may refer.

It is not proposed to offer a complete treatise on Pathological Anatomy, nor on Medical Jurisprudence, but rather a work which bears the same relation to them that a dissecting manual does to a complete Anatomy.

The book is divided into four parts. The first gives the method of performing autopsies on the bodies of adults and of young children. The second gives in detail the lesions which have been observed in each organ of the body. The third gives the lesions which are found after deaths from general diseases, from violence, and from poisons. The fourth gives a short classification of tumors.

FRANCIS DELAFIELD,

12 West Thirty-second street.

JUNE, 1872.

PART I.

ON THE METHOD

OF MAKING

POST-MORTEM EXAMINATIONS.

ON THE METHOD

OF MAKING

POST-MORTEM EXAMINATIONS.



THE particular object of making a post-mortem examination varies in different cases. It may be to determine whether a person has died from violence or poisoning ; to account for a sudden death ; or to study the lesions of disease. In any case, the examination should embrace the entire body, not merely the suspected organ, and it should be recorded at the time it is made.

It is necessary to be very careful in asserting that a given lesion is or is not a sufficient cause for death. The causes of death, especially of sudden death, are often very obscure. Persons who have been suffering from chronic diseases frequently die suddenly without any discoverable cause. At all periods of life, from infancy to old age, we meet with cases of sudden death which we are unable to account for by the clinical history or the post-mortem examination. In such cases, it is better to avow our ignorance than to attribute the death to a congestion of the brain or a disease of the kidneys—which does not exist.

EXTERNAL INSPECTION.

Before commencing the examination of the internal viscera, it is always necessary to make some inspection of the external surface of the body. The minuteness of this inspection will depend upon the character of the case. In the case of an un-

known person, or of one who is supposed to have died from unnatural causes, it is necessary to search for and record not only all contusions, wounds, etc., their size and situation, but also any physical peculiarities of hair, eyes, moles, etc., by which the person may be identified. In ordinary examinations, we only look for the evidences of external injuries, for skin diseases, ulcers, etc., for œdema, gouty deposits, abscesses, enlarged lymphatic glands, etc. The glans penis and prepuce are to be carefully searched for syphilitic cicatrices.

It is customary to find certain changes in the external appearance of the body, which are due to the cessation of vitality in the tissues and to the commencement of decomposition. I speak now of bodies which have not yet been buried, and which have been kept in the ordinary way, partly covered by a shroud, and lying on their backs in a loosely-covered coffin. If the bodies have been left in their ordinary clothes, the appearances are much the same.

In such bodies, one of the first noticeable changes is the paleness of the skin and its mottling with irregular livid patches. After a short time, the blood settles in the vessels of the more dependent portions of the body, and the skin which covers the back of the trunk and extremities becomes of a livid red color. In many cases, if we cut through the skin, we find the tissues beneath congested and infiltrated with bloody serum. In bodies which have been kept for a number of days in cold weather, this red color of the skin is also seen on the anterior portions of the body, especially the face and neck. In hot weather, the red color is very soon altered by decomposition. If the epidermis has been detached at any point, the skin beneath this is dry, hard, and red.

In warm weather, we may find, a few hours after death, broad, bluish lines, corresponding to the cutaneous veins, ramifying in the skin of the neck and thorax. These lines are formed by the escape of the coloring matter of the blood from the vessels.

Within a few hours after death, even in cold weather, there is usually some escape of frothy blood and mucus from the mouth and nose.

If the eyelids are not closed, the conjunctiva and cornea soon become dry, brown, and hard. The eyeballs also become flaccid.

After a considerable length of time, the skin of the abdomen becomes green. Still later, decomposition fairly sets in.

The entire body is of a dark green color. The tissues are infiltrated with serum. The abdomen is distended with gas. Then the color changes from a green to a reddish brown. The epidermis is detached. The skin is covered with maggots. The entire body is swollen from the formation of gases. The face can hardly be recognized. The nails drop off, and the scalp becomes detached. When a body is in this condition, it can hardly be determined whether one month or five months have elapsed since death.

After this, all the soft parts change into a formless, putrescent mass. The cavities are open, the viscera are indistinguishable, the bones are left bare.

The rapidity with which these changes take place varies under the influence of a great number of conditions.

The bodies of infants usually decompose more rapidly than those of adults. Fat bodies putrefy more quickly than lean ones. The bodies of persons who die suddenly from violence decompose less rapidly than the average, unless the body is a good deal mangled. Exhausting diseases, fevers, and the puerperal condition are followed by rapid decomposition, as is also death from suffocating gases. Poisoning by alcohol, by arsenic, and by sulphuric acid may preserve the bodies for an unusual length of time. Atmospheric air, moisture, and warmth quicken decomposition. At the same temperature, a body which has been for one week in the air, one which has been two weeks in water, and one which has been eight weeks buried in the usual way, will all exhibit the same degree of decomposition.

The Rigor Mortis.—It is proper to notice whether or not the body is in the condition of post-mortem rigidity. More attention has, perhaps, been given to this particular post-mortem condition than it well deserves.

According to Kühne, the rigor mortis is produced by a change in the muscular fibres. The fibres first lose their contractility,

then there is coagulation of the myosine, and loss of elasticity; then the muscular tissue becomes acid. When this acidity has reached its height, the muscle becomes softer, and the rigor mortis gradually disappears. Finally the acid condition is succeeded by an alkaline fermentation, and decomposition ensues.

The rigor mortis generally begins in the muscles of the lower jaw and back of the neck; it then extends to those of the face, neck, thorax, arms, and finally the legs. It usually disappears in the same order.

It usually begins in from 8 to 20 hours after death, but often much sooner. The bodies of persons killed on the field of battle, and of those who have been drowned, sometimes seem to be overtaken by the rigor mortis at the very instant of death. The bodies retain the same position and the face the same expression which they had in the last moments of life.

The rigor mortis may continue for from one to nine days.

Generally, but not always, death from narcotic poisons is followed by a feeble and short rigidity.

It is said that death by lightning is followed by rapid and intense rigidity.

Casper says that in foetuses born before term he has never observed any rigidity, and that in young children it is feeble and of short duration.

It is asserted by some and denied by others that death from suffocation is followed by a short and feeble rigidity.

The degree and duration of rigor mortis after death from violence, from different diseases, etc., is stated so variously and so contradictorily by different observers, that no definite rules can be given concerning it.

The temperature of the living body is 98° to 99° F. In consequence of disease, the temperature may be increased several degrees. After death, the body gradually cools to the same point as the surrounding air. This is said to take place in from fifteen to twenty hours. Taylor, from the examination of one hundred bodies, states that the average heat of the skin of the abdomen at a period of two to three hours after death is 77°; at four to six hours, 74°; at six to eight hours,

70°; at twelve hours, 69°. The internal viscera retain their heat longer than the surface of the body.

It is said that, after sudden death from accidents, apoplexy, acute disease, and asphyxia, the body retains its heat for an unusually long period.

It is both asserted and denied that after death from hæmorrhage the body cools rapidly. The body of an adult cools more slowly than that of a child or of an old person; that of a fat person more slowly than that of a lean one. The more the body is covered up, the less rapidly it cools.

In some cases, there is an exceptional retention and even an increase of heat in the dead body. Dr. John Davy reports that in a case of death from rheumatism, after the viscera had been exposed for several minutes, the temperature of the left ventricle of the heart was 113°, and that of the liver 112°. In a second case, six hours after death, the temperature of the heart was 108°.

It is stated that after death from yellow-fever and cholera the temperature increases for several hours after death.

There are also recorded a number of cases in which the body retained its heat for several days without known cause.

It will be seen, from what has been said, that if we are called upon to pronounce upon the length of time which has elapsed since death in a given case, this is only to be done approximately and probably. It is necessary to take into consideration the cause and manner of death, the condition of the individual, the state of the atmosphere, the manner in which the body has been kept after death. And even after making these allowances, we can only say that probably a person has been dead for such or such a time.

Contusions.—It is often important to determine whether violence has been inflicted on a given body before death. With reference to this point, we must remember in the first place that blows and falls of sufficient violence to fracture the bones and rupture the viscera may leave no marks on the skin, even if the individual has survived for several days.

In the second place, there are a number of post-mortem appearances which simulate ante-mortem bruises. If we observe

a severe contusion, as it exists during life, we see that the injury at first leaves no mark, or only a general redness. After a short time, the injured part is swollen and of a red color. This color is succeeded by a black and blue, and this again gradually fades out into a yellow. All these changes are due to an escape of blood from the vessels, and to subsequent changes in its coloring matter. If, therefore, we cut into such an ecchymosis after death, we shall find extravasated blood, or the coloring matter of the blood in the form of granular pigment in the tissues. Post-mortem discolorations, on the other hand, although their external appearance may resemble that of ante-mortem ecchymosis, are not formed by an extravasation of blood, but by a circumscribed congestion of the vessels, or by an escape of blood-stained serum. If we cut into such discolorations, therefore, we find no blood outside of the vessels.

An abrasion of the skin, by which the epidermis has been rubbed off, presents the same appearance whether produced before or after death. In either case, the denuded cutis becomes hard, dry, and parchment-like.

Blows on the skin of a body which has not been dead for more than two hours may produce true ecchymosis, with extravasation of blood, such as can hardly be distinguished from those formed during life.

Wounds.—We should notice the situation, extent, and direction of a wound; the condition of its edges and of the surrounding tissues. If it is a deep penetrating wound, a probe or director should be passed into it before it is laid open.

If we find the edges of a wound inflamed and suppurating, or commencing to cicatrize, it must have been inflicted some time before death. If a wound has been inflicted a short time before death, its edges are everted; there is extravasation of blood into the surrounding tissues, the vessels contain coagula, or all these signs may be absent. The chief characters of a wound inflicted after death are absence of bleeding, non-retraction of the edges of the wound, and no extravasation of blood. A wound inflicted within two hours after death resembles more closely one inflicted during life. In general, unless a wound is old enough for its edges to present inflammatory

changes, we must be very careful in asserting its ante-mortem or post-mortem character.

Fractures.—It may be important to determine whether a bone was fractured before or after death. This point cannot always be decided. Generally a much greater degree of force is necessary to fracture bones in the dead than in the living body. Fractures inflicted during life are, as a rule, attended with more extravasation of blood and evidences of reaction in the surrounding tissues. But fractures inflicted within a few hours after death may resemble these very closely.

Scars.—The presence of cicatrices and their appearance should be noticed. Scars produced by an actual loss of substance, or by a granulating wound, never disappear entirely. Slight and superficial wounds, however, produce marks which are not permanent. The discolorations produced by tattooing may fade away during life. Not infrequently tattoo marks, which were observed during life, can no longer be found after death.

INTERNAL EXAMINATION.

In making the internal examination, it should be our object to make it both as thoroughly and as rapidly as possible. In order to do this, we must follow a regular method. It is always necessary to examine not merely the particular organs in which we expect to find lesions, but also all the other viscera. If this is not done, we are always liable to fall into grave errors.

After completing the external inspection of the body, we commence the internal examination. This is best done in the following order :

The Head.—The scalp is divided by an incision across the vertex from ear to ear. The flaps are directed forward and backward, taking up the temporal muscles with the skin and leaving the pericranium attached to the bone. The internal surface of the scalp and the pericranium are to be examined for ecchymoses and inflammatory lesions. A circular incision is then made with the saw and the roof of the cranium removed. The incision in front should pass through a point three and a half inches above the root of the nose, behind through the occipital protuberance.

If fracture of the cranium is suspected, the bones should be sawn entirely through. If not, it is sufficient to saw partly through and then separate the calvarium with the chisel. When the roof of the cranium is thus entirely loosened, a large hook is introduced under the upper edge of the calvarium, and this is wrenched off with a sudden jerk. Sometimes the dura mater is so adherent to the roof of the skull that the latter cannot be torn from it. We must then cut through the dura mater at the level of the incision in the bones, and take out the brain with the calvarium.

When the calvarium is removed, we notice whether or not it is symmetrical. The cranial bones normally increase in size by a growth of bone at the edges of the sutures. If any suture becomes completely ossified and closed before the others, the bones will be unequally developed. Thus an early closure of the sagittal suture produces a long, narrow cranium, etc.

The thickness of the cranium and its density vary very considerably within the limits of health. There are often deep depressions on the inner surface of the skull along the sagittal suture, caused by the pressure of the pacchionian bodies.

We observe the degree of congestion of the bone, the existence of fractures and of inflammatory lesions.

The Dura Mater is now exposed to view. It will be found more or less adherent to the calvarium. A moderate degree of adherence, especially in old persons, does not denote disease. Very extensive and firm adhesions are usually produced by inflammation. Near the median line the pacchionian bodies often project through the dura mater. We must look for clots, for new growths, and for inflammatory lesions on the outer surface of the dura mater. The longitudinal sinus should be laid open with the scissors, and its contents examined. A circular incision is then made through the dura mater, in a line corresponding with the incision through the skull, the falx is entirely divided between the anterior lobes of the cerebrum, and the entire membrane drawn back. We notice the existence of adhesions to the pia mater, of clots, of the products of inflammation, and of new growths.

The Pia Mater is now exposed, covering the convex surface of

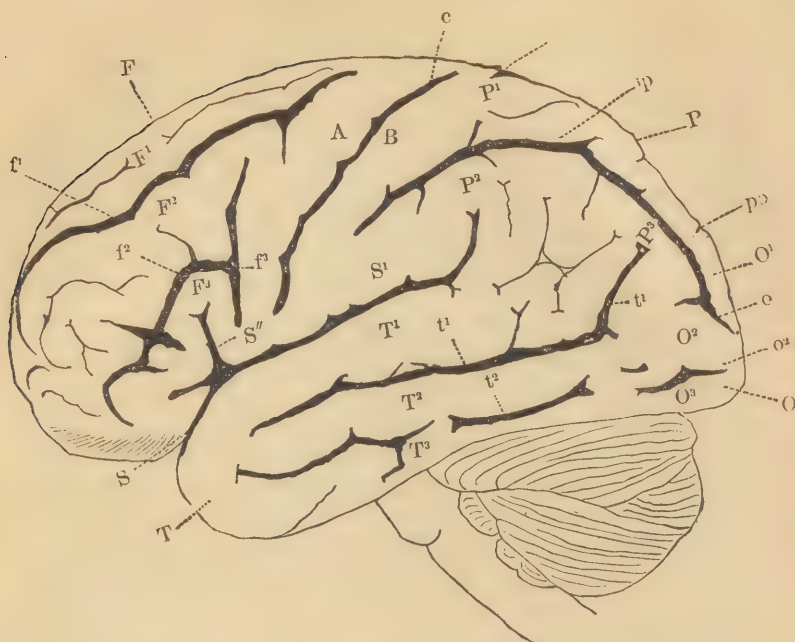
the brain. The degree of congestion and the existence of serum, pus, or blood beneath or in the pia mater, are to be ascertained at this time, before the brain is removed. The pia mater in old persons loses its transparency and becomes thick and white; this change is most marked along the longitudinal fissure and the large vessels. It is a simple senile change, but is frequently mistaken for an inflammatory lesion. Marked and general thickening of the pia mater, however, is produced by chronic inflammation. Along the longitudinal fissure, and sometimes at a distance from it, we find little whitish, wart-like bodies. These are the pacchionian bodies, which are found normally in adult life.

The amount of serum beneath the pia mater varies. A considerable amount, especially in cachectic persons, may exist without brain disease. Clear serum lifting up the pia mater and separating the convolutions of the brain may be simply dropsical or due to chronic meningitis. Turbid and purulent serum beneath and in the pia mater are produced by acute meningitis.

The Brain.—After examining the convex surface of the brain, the anterior lobes of the cerebrum are to be pulled gently backward, the cranial nerves and the tentorium divided, the medulla oblongata cut across as low down as possible, and the brain thus removed from the cranium.

The adult brain weighs, in the male, from 46 to 53 oz.; in the female, from 41 to 47 oz. Besides the lobes and fissures of the brain, it is important to notice the convolutions and sulci, in order to note the exact situation of any lesion.

The brain is first laid on its convex surface, and the anterior, middle, and posterior cerebral arteries, the basilar, and the carotids examined for emboli, thrombi, and atheroma. Extravasations of blood, new growths, and inflammatory lesions are also to be looked for. The brain is then turned over on its base. An incision is made through the pia mater, over the convex surface of the cerebrum; this membrane is stripped up, and its adherence to the brain, its thickness and general appearance are noticed. The two halves of the cerebrum are then to be gently separated until the superior surface of the corpus callosum is



SIDE VIEW OF THE BRAIN.

F. Anterior lobe.
O. Posterior lobe.
T. Middle lobe.

S. Fissure of Sylvius.
S'. Horizontal branch.
S''. Vertical branch.

C. Sulcus centralis, or fissure
of Rolando.
A. Anterior cent. convolution.
B. Posterior cent. convolution.

F¹, F², F³. First, Second, and Third Frontal Convolutions.

f¹, f², f³. Superior, Inferior, and Vertical Frontal Sulci.

P¹. Postero-parietal Lobule. P². Inferior Parietal Lobule. P³. Angular Gyrus.

i p. Inter-parietal Sulcus. p o. Parieto-occipital Fissure.

O¹, O², O³. First, Second, and Third Occipital Convolutions.

o¹. Transverse Occipital Sulcus. o². Inferior, Longitudinal, Occipital Sulcus.

T¹, T², T³. First, Second, and Third Temporal Convolutions.

t¹ t². First and Second Temporal Sulci.

exposed. An incision is then made through the junction of the corpus callosum and cerebrum, cutting outward and downward, into each lateral ventricle. This incision should be made carefully, so as not to cut through the ventricles into the ganglia below. The incision, thus made through the roof of each ventricle, is prolonged backward and forward, in the direction of the cornua, until the entire cavity of each lateral ventricle is exposed. A long incision outward and downward is then made into each half of the cerebrum, from the outer edge of

the lateral ventricles, nearly to the pia mater. A second incision is then made from this cut surface outward, and this is repeated until the cerebrum is divided into a number of long prismatic-shaped pieces held together by the pia mater. The velum interpositum is then dissected up, and the fourth ventricle opened. To complete the examination, cross-sections are made through all the large ganglia, the medulla, and the cerebellum. Care must be taken not to overlook small clots in the medulla oblongata.

The Base of the Cranium.—We now return to the skull. The remaining sinuses of the dura mater should be opened. This membrane is then to be entirely stripped off from the bone. The bone is to be examined for fractures, inflammatory lesions, and new growths.

The Eyes can be removed by comminuting the roof of the orbit with a hammer, removing the fragments of bone, dissecting away the fat and muscles, so as to expose the optic nerve and posterior half of the eye. With a pair of scissors we can then cut away that portion of the globe which is not covered by conjunctiva, and remove it with the optic nerve. The anterior portion of the globe with the cornea is left in place. The nerve and portion of the eye removed are to be placed at once in Müller's fluid.

The Spinal Cord.—The examination of the spinal cord may, in many cases, be dispensed with. When it is necessary, it is most conveniently performed after the removal of the brain.

The body should be placed face downward, with a block under the thorax, and the head dropping over the edge of the table. An incision is made through the skin and muscles along the entire length of the spine, and the soft parts dissected off on each side, so as to expose the laminæ of the vertebral column. The laminæ are then divided close to the articulating processes, with the rachitome, the saw and chisel, with a sharp chisel, or with strong cutting forceps. The spinous processes and laminæ are then torn away together, and the cord exposed. The membranes of the cord may be found very considerably congested without indicating the preëxistence of any disease. The roots of the spinal nerves are then to be cut across, and

the cord removed in its membranes, care being taken not to press it in any way. It is the safest plan not to grasp the cord itself, but with a forceps to seize the dura mater, and thus lift up the cord. The dura mater is then laid open with scissors over the entire length of the cord.

If disease of the cord is suspected, and no lesions are to be seen with the naked eye, the cord should always be preserved for microscopical examination. For this purpose, it should be cut into pieces an inch long; these pieces are then loosely tied in their order with a cord, and suspended in a jar containing a solution of Potass. Bichrom., gr. xv. to $\frac{3}{2}$ of water. After remaining in this for 10 days, the cord should be removed to a solution of Ac. Chromic, gr. ij to $\frac{3}{4}$ of water, and remain in this until hard enough to be cut into thin sections.

After the removal of the cord, fractures and displacements of the vertebræ are easily recognized.

The Thorax and Abdomen.—To examine these cavities the body is replaced on its back. A single straight incision is made from the top of the sternum to the pubes. For this purpose, a large scalpel should be used, and the first incision should divide every thing down to the sternum and peritoneum. A short incision should then be made through the peritoneum; into this opening two fingers of the left hand are introduced, and the peritoneum divided from the ensiform cartilage to the pubes. The skin and muscles are then dissected off from the thorax on both sides as far back as the false ribs. In doing this, we notice the amount of subcutaneous fat and the condition of the muscles.

The abdominal cavity is now exposed, and we have first to notice the position of the viscera.

The Liver is situated in the right hypochondriac region, filling up the concavity of the diaphragm. Its upper border reaches in the linea mammalis to the fifth intercostal space; in the linea axillaris, to the seventh intercostal space; close to the vertebral column, to the tenth intercostal space. At the median line the upper border of the liver corresponds to the lower border of the heart. The left lobe extends about three inches to the left of the median line. The lower border of the

* note position of diaphragm

right lobe usually reaches to the free border of the ribs, while the left lobe is visible for about an inch below the ensiform cartilage. In women, the liver is usually lower than in men.

The position of the liver is altered by changes in the thoracic cavity, forcing it downward; by changes in the abdominal cavity, forcing it upward; by constriction of the waist by tight lacing, forcing it either downward or upward; by changes in the size of the organ itself.

The liver may be not only displaced downward, but dislocated so that its convex surface faces the abdominal wall, and its posterior edge faces upward against the diaphragm.

The Stomach is situated in the left hypochondriac and epigastric regions, extending also into the right hypochondrium. It lies in part against the anterior wall of the abdomen, and in part beneath the liver and diaphragm, and above the transverse colon. Its anterior surface, which is directed upward and forward, is in contact above with the diaphragm and the under surface of the liver, and lower down with the abdominal wall, opposite to the epigastric region. Its posterior surface is turned downward and backward, and rests upon the transverse mesocolon, the pancreas and the great vessels. The lesser curvature or upper border has attached to it the gastro-phrenic ligament and gastro-hepatic omentum. To the greater curvature, or lower border, is attached the gastro-colic omentum. Its cardiac orifice terminates in the œsophagus, its pyloric end in the duodenum.

When the stomach is distended, the greater curvature is elevated and carried forward, the anterior surface is turned upward, and the posterior surface downward.

The organ when distended with food or gas, is prominent; when empty, may be hardly seen; when the intestines are dilated, may be entirely covered by them.

The Transverse Colon passes from one side of the abdomen to the other through the upper part of the umbilical region. It may be lower than the umbilicus, or high up against the liver and diaphragm. It may be distended with gas or contracted.

The Omentum should be found smoothly spread over the

surface of the small intestines, but may be rolled up and displaced in a variety of positions, or may be adherent at some point.

It is convenient at this stage of the examination to look over the peritoneal cavity for serum, or inflammatory lesions, and to notice the existence of invagination, incarceration, or herniæ of the intestines.

If the existence of air or gas in the pleural cavities is suspected, the abdominal cavity should be filled with water and the diaphragm punctured below the level of the fluid. If any air be present, it will escape in bubbles through the water.

The Thorax.—We now leave the abdominal viscera in place, and proceed to the examination of the thorax.

With a costatome, or a strong knife, the costal cartilages are divided close to the ribs, the clavicles are disarticulated from the sternum, and the latter removed, taking care not to wound the large veins.

We first examine the position of the heart and lungs.

The Heart, covered with the pericardium, extends vertically from the second intercostal space to the sixth cartilage, and transversely from a little within the left nipple to about half an inch to the right of the sternum. The apex of the heart is opposite to the fifth intercostal space, about $3\frac{1}{2}$ inches from the middle of the sternum. From the fifth intercostal space to the upper edge of the fourth cartilage, the heart is covered by the lungs. The portion of the heart uncovered by the lungs is of an irregularly triangular shape. The vertical side of the triangle is 2 inches, the horizontal $2\frac{1}{2}$, and the oblique 3 inches in length. The area of the heart which is found uncovered will vary very much according to the degree in which the lungs collapse after death. Any disease which diminishes the size of the lungs, or any pleuritic adhesions which retract or bind down the lungs, may increase the area of exposed heart. On the other hand, emphysema, pneumonia, or any disease which increases the size of, or retains the air in, the lungs, may diminish the area of exposed heart. The pericardium is now opened by a vertical incision on its anterior surface. The existence of serous, fibrinous, or purulent exudation, and of adhesions, are

to be noticed. A small amount of clear serum exists normally in the pericardial sac, and this serum may be blood-stained from commencing decomposition. White thickenings of the pericardium on the anterior surface of the right ventricle are often seen; they do not indicate disease.

Now that the pericardial sac is opened, the position of the heart can be clearly seen. It lies transversely in the chest, its long axis at an angle of 60° with that of the thorax. The portion of the heart which is first seen is the anterior surface of the right ventricle; upward and to the right of this is the right auricle, which lies $\frac{2}{3}$ on the right side of the sternum, and $\frac{1}{3}$ behind it. Its upper border usually corresponds with the plane of the middle of the anterior end of the second intercostal space on the right side. Its size varies with the amount of blood it happens to contain. The left auricle lies behind the root of the pulmonary artery, so that only its appendix is visible. The middle of the auricle corresponds to the third costal cartilage. Of the left ventricle only a narrow rim is seen on the left side of the right ventricle.

The opening of the tricuspid valve corresponds to the sternal end of the fourth cartilage, behind the left half of the sternum. The pulmonary valve is behind the left edge of the sternum, at the middle of the anterior end of the second intercostal space.

The mitral valve is just above the upper edge of the third left cartilage, near the left edge of the sternum, behind and below the edges of the pulmonary valves.

The aortic valve is just below the sternal end of the third left cartilage.

The heart should now be removed unopened, by cutting through the vessels at its base, after first passing the hand over the arch of the aorta to ascertain whether any aneurism is present. To determine the sufficiency of the aortic and pulmonary valves, a full stream of water is poured into the aorta and pulmonary artery, and we observe how well the valves support the column of fluid. To ascertain the sufficiency of the mitral and tricuspid valves, the auricles are first laid open so as to expose the upper surfaces of the valves. A large pipe is passed through the aorta or pulmonary artery beyond their valves, and

a moderate stream of water allowed to flow into the ventricles. The auriculo-ventricular valves will be swollen outward, and we can observe the degree of their sufficiency. The tricuspid valve is normally somewhat insufficient.

To ascertain the size of the different valvular openings, we introduce one or more fingers, held flat and with their edges in contact, into each of the orifices, and then measure the width of the fingers at the point where they fill the orifice. In this way we find that the aortic valve measures about 1 inch; the mitral valve, $1\frac{3}{4}$ inches; the pulmonary valve, $1\frac{1}{4}$ inches; the tricuspid valve, 2 inches.

In order to examine the interior of the heart, we first make an incision through the anterior wall of the right ventricle, close to the septum, and reaching to the apex of the ventricle; through this opening the blade of the enterotome is passed into the pulmonary artery, and the ventricle and artery laid open. With a little care, the incision may be made to pass through one of the points of junction of the pulmonary valves. The ventricle may be empty, or contain fluid blood, or red clots, or succulent yellow clots, or firm whitish clots, either free or attached to the heart-wall. The succulent yellow clots are very common; they are sometimes intimately entangled with the trabeculæ of the ventricular wall; they are not a pathological condition, but are found in persons dying in every way; they are formed after death or during the last moments of life. The condition of the pulmonary valves, of the endocardium, and the thickness and appearance of the wall of the ventricle are then noticed.

The left ventricle is then opened and examined in the same way, by an incision through its anterior wall close to the septum. We sometimes see the endocardium of the upper part of the left ventricle thick and white, without the existence of valvular lesions, or any clinical history of disease. To complete the examination of the cavities, the enterotome is passed into each ventricle and auricle, and an incision made from the apex of each ventricle through its outer border, and the outer border of the corresponding auricle. In this way the auriculo-ventricular valves are fully displayed.

After removing the blood, the heart should finally be weighed. In adults, the normal average weight of the heart is, in males, 10 oz.; in females, 8 oz. The average relative weight to that of the body is, in males, 1 : 158; in females, 1 : 149. According to Bizot, the thickness of the wall of the left ventricle is, in males, 7-16 in.; in females, 5-16 in.; of the right ventricle, in males, 3-16 in.; in females, 2-16 in.

	Males.	Females.
The diameter in French lines of the mitral valve is	45 17-30	41 1-3
“ “ “ tricuspid valve is	54 12-61	48 1-4
“ “ “ aortic valve is	31 15-61	28 4-5
“ “ “ pulmonary valve is	32 21-61	30 7-60

Generally speaking, the size of the heart corresponds to the size and development of the individual. In judging of an increase or decrease in its size, we must consider the weight of the organ and the thickness of its walls. If the patient dies while the heart is contracted, the walls of the ventricle will appear thicker, their cavities smaller than usual. If the patient die of some exhausting disease, like typhoid fever, or if decomposition has commenced, the heart-walls will be flabby, and the cavities will appear larger than usual.

The Pleural Cavities are now examined. The hand is passed into each, and the existence of serous or fibrinous exudation, or of old adhesions, are ascertained. The method of ascertaining the presence of air has already been given.

The Lungs.—Each lung is now lifted up in turn, the vessels, etc., at its base divided, and the organ removed. If the pleura is very adherent, it is better to strip off the costal pleura with the lung. After inspecting the external surface of the lung, we proceed to open the bronchi. For this purpose, we use a pair of scissors with long, narrow, blunt-pointed blades, one blade a little longer than the other. The lung is held in the left hand, with its base upward. We first open the large bronchi which run on the inner side of the lower lobe; then the other bronchi of the lower lobe; and afterward those of the upper lobe. Each bronchus should be followed to its termination, and then its different branches opened.

In the larger and middle-sized bronchi, the cartilage in their

walls does not form a continuous coat, but appears like irregular white patches, visible from the internal surface of the bronchi. This appearance must not be mistaken for a morbid change.

In bodies which have been dead for several days, especially in cold weather, the bronchial mucous membrane may be red and swollen as a post-mortem change.

The contents of the stomach sometimes find their way after death into the pharynx, and then down into the trachea and bronchi, giving to them a peculiar brownish or even gangrenous appearance.

Bronchitis which has existed during life does not always leave lesions which can be appreciated after death.

After the examination of the bronchi, the lung is turned over, the vessels, etc., at its root grasped with the left hand, and a long, deep incision made from the apex to the base. We observe the appearance and texture of the lungs, whether the air-cells are dilated, (emphysematous,) or filled with serum, blood, or inflammatory exudation. All fluids can be pressed out of the air-cells without breaking down the lung tissue. Solid inflammatory exudation, on the other hand, renders the lung more resistant and easily broken down. It is the rule to find the lower lobes much more congested than the upper.

To examine the lungs microscopically, we make sections of the fresh lung with a double-bladed knife; or small pieces of the lung are placed in alcohol, (95 per cent,) and kept there, changing the alcohol from time to time, until they are hard enough to be cut with a razor.

The Pharynx, Larynx, and Esophagus.—If these organs are to be examined, we prolong the incision in the thorax to a point one inch below the chin. The soft parts are dissected off from the larynx, taking care not to cut the thyroid body, an incision is made through the floor of the mouth, following the internal surface of the inferior maxilla. Through this incision, the fingers are introduced into the mouth, the tongue drawn down, the posterior wall of the pharynx divided above the tonsils, and the pharynx and larynx thus drawn out together. These organs are then seized with the left hand and dragged downward, and, with the aid of the knife, the entire length of the trachea

and œsophagus are torn out, cutting the œsophagus just above the stomach. If the contents of the stomach are to be preserved, a ligature is placed around the œsophagus just below the point at which it is cut off.

We now with the enterotome slit up the pharynx and œsophagus on their posterior surfaces. The mucous membrane thus exposed is examined for evidences of caustic poisons, of inflammation, of new growths, strictures, etc.

The enterotome is then introduced into the larynx, and this organ and the trachea are laid open by an incision through their posterior walls. Here we look for œdema of the aryteno-epiglottidean folds, (œdema glottidis,) and for the evidences of catarrhal, croupous, ulcerative, and syphilitic inflammation. New growths of various kinds, and diseases of the laryngeal cartillages are also met with. Œdema and redness of the larynx may be produced by post-mortem changes, especially in bodies which have been kept for several days in cold weather.

To preserve the larynx and trachea for microscopical examination, I have obtained the best results by suspending them in a jar, containing equal parts of alcohol and of a solution of Potass. Bichrom., gr. xv. to the $\frac{3}{4}$ 1 of water. The proportion of alcohol is to be afterward increased until the mucous membrane is hard enough to be cut into thin sections.

The Abdomen.—We now return to the abdominal cavity. We first dissect off the omentum. If tubercles of the peritoneum exist, they are best seen and studied in the omentum.

The colon is then raised up and dissected free, to the cœcum on one side, and to the rectum on the other. The colon and small intestines are then drawn first to the right and then to the left side, so as to expose in turn the left and right kidneys. As each kidney is brought into view, an incision is made through the peritoneum over the track of the ureter. The ureter is then followed out from the kidney to the bladder and its condition ascertained.

The Kidneys are then removed, separating the peritoneum and fat from them with the hand and dividing the vessels with the knife. At the same time we remove the suprarenal capsules, which are attached to the upper end of each kidney.

Venous Plan of examination

- 1 - omentum
- 2 - cœcum
- 3 - left kidney, suprarenal capsule & ureter
- 4 - right
- 5 - bladder, prostate gland, vesicular seminae & urethra

Each kidney in turn is then taken up. An incision through the capsule is made along the convex edge of the kidney, and this membrane stripped off from it. We notice the degree of adherence of the capsule to the kidney, and also the surface of the latter, whether smooth or roughened, pale or congested. An incision is then made along the convex surface down to the pelvis, so that the organ is divided into two halves. We observe the relative thickness of the cortical and pyramidal portions, as well as the size of the entire organ. To ascertain the latter point, it is well to weigh each kidney: the normal weight is from $4\frac{1}{2}$ to 5 oz. It is necessary to remember, however, that in a kidney which is a good deal atrophied there may be an increase of fat in the pelvis, which gives the organ nearly its normal size and weight, while the kidney tissue proper has really in great measure disappeared.

We now inspect more closely the kidney tissue, especially the cortical portion. It is well known that the pyramids consist of tubes running nearly in straight lines from the apex to the base of each pyramid. These straight tubes pass from the pyramids into the cortex in bundles, retaining their straight course until they nearly reach the surface of the kidney, then bending over on each side, become convoluted and end in malpighian bodies. In this way, the cortex of the kidney is divided up into alternate bands of straight and convoluted tubes, both sets of bands being perpendicular to the surface of the kidney. About the convoluted tubes is a rich venous plexus, and as the blood usually remains after death in this plexus and in the malpighian bodies, the bands containing the convoluted tubes appear red, while those containing the straight tubes are grayish white. In a normal kidney, therefore, the cortex should be regularly striped in red and white bands. If there be extensive congestion, the entire cortex is red. If the epithelium of the tubes degenerates and fills them up, or if there are changes in the interstitial tissue, the regular bands are lost and the cortex is irregularly mottled. If the tubal epithelium becomes filled with oil globules, this is indicated by an opaque yellow color of the parts affected. In many cases, therefore, the existence of kidney disease can be recognized with the naked eye. To deter-

6-(a) - testicles, spermatic cord & vas
 (b) - vagina, uterus, fallopian tubes
 ovaries, parametria
 7 - rectum
 8 - duodenum & jejunum & ileum
 (b) - appendix vermiformis

mine the presence of waxy degeneration, the cut surface of the kidney is washed with water to get rid of the blood, and then brushed repeatedly with an aqueous solution of Iodine. The malpighian bodies and blood-vessels are the parts usually affected by the degeneration. If they are so affected, they will appear as red dots and lines on a yellow ground. Sometimes a whitish fluid is seen in the pelvis and can be squeezed from the papillæ: this is produced by a post-mortem desquamation of the epithelium.

If there be any doubt as to the condition of the kidneys, microscopical examination is necessary. For this purpose, we make sections of the fresh kidney with a double-bladed knife, or the organ is frozen and sections made with a razor, or it may be preserved and hardened in a solution of Potass. Bichromate, or in alcohol. The condition of the tubal epithelium is best seen in fresh sections, or in sections from specimens hardened in alcohol and examined in glycerine; that of the interstitial tissue in Bichromate or alcohol preparations stained with carmine. Sections are absolutely necessary even to ascertain the condition of the epithelium. For not only is the epithelium of the tubes in the pyramids different from that of those in the cortex, but in the cortex the epithelium of the straight tubes differs from that of the convoluted tubes. In the straight tubes, the epithelium is regular, the edges of each cell and its nucleus well marked, and its body only slightly granular. In the convoluted tubes, the epithelium looks like a mass of granular matter, in which nuclei are imbedded at regular intervals.

The Suprarenal Capsules are, in the foetus of an ovoid, in the adult, of a triangular shape. They are situated at the upper and inner border of the kidney, to which they are loosely or closely fitted. Their size varies. In the adult, the transverse diameter is from 40 to 55 mm.; the vertical, 20 to 35 mm.; their thickness is from 2 to 6 mm. They weigh from 8 to 120 grains. In new-born children, their weight is to that of the kidney as 1.3; in the adult, as 1.28.

They are composed of a cortical and a medullary portion, the cortex forming a shell around the medula. They are inclosed

- 9- stomach
 10- hepato-duodenal ligament, gall-duct,
 and portal, gall-bladder, liver.
 11- pancreas, celiac ganglion.
 12- mesentery, glands mesenteric

in a fibrous capsule, from which fibrous processes extend into the cortex.

The cortex consists of two layers : an outer yellow layer, and an inner thin, reddish-brown layer. Next to the capsule is a thin layer of polygonal and rounded nucleated cells, arranged in rounded groups. To the inside of this is a broad layer of polygonal, cuboid, or rounded nucleated cells, arranged in parallel rows or in long follicles. The long axes of the rows, or follicles, are at right angles to the surface of the organ. The inner layer of the cortex is composed of the same cells closely packed in a stroma with fine meshes.

The medullary portion consists of a delicate fibrous reticulum, in the meshes of which are cells. The cells are very fragile, of polygonal or stellate shape. They are arranged in groups, or are irregularly contained in the stroma.

The blood-vessels are very numerous ; they are derived from the aorta, the phrenic, the cœliac, and the renal arteries.

The nerves are also very abundant. They are derived from the semi-lunar ganglion, the plexus renalis, the phrenic, and the vagus. They are especially numerous in the medullary portion. Ganglion cells are found in the course of the nerves in the medullary portion.

It is very common to find the suprarenal capsules changed by decomposition. The inner layer of the cortex softens and breaks down, so that the outer layer of the cortex forms a sort of cyst filled with a reddish-brown, broken-down material. The cells of the cortex usually show a considerable degree of fatty degeneration.

The Spleen is next to be examined. It is an organ of elliptical, flattened shape, slightly curved on its flat surface. It is situated on the left side of the stomach, with its long axis vertical. Its convex surface is in contact with the diaphragm ; from its concave surface, or hilus, its vessels are given off. Its long diameter reaches from the seventh intercostal space to the eleventh rib. Its upper portion is separated from the ribs by the lungs ; its lower portion by the diaphragm. Its normal length is from $4\frac{3}{4}$ to $5\frac{1}{2}$ inches ; its breadth from $3\frac{1}{4}$ to 4 inches ; its thickness, $1\frac{1}{4}$ to $1\frac{3}{4}$ inches. It weighs from 8 to

13 - small along intestine
14 - adno-peritoneal glands, receptaculum
chyl, aorta, vena cava inferior

10½ ounces. But these measurements and weights may vary considerably within the limits of health. The spleen is inclosed in a fibrous capsule covered with peritoneum. The parenchyma is made up of a stroma formed of blood-vessels and fibrous tissue, and of a soft, reddish pulp, in which are imbedded white, rounded bodies—the malpighian bodies. In the human spleen, the malpighian bodies are usually hardly perceptible to the naked eye. Sometimes they are very plain. Sometimes the fibrous stroma is very apparent; sometimes not.

The size, consistence, and color of the organ may vary a good deal without any known cause. Decomposition softens it. Thickenings of the capsule and adhesions are very common.

The Intestines.—If the intestines require careful examination, we divide the rectum, seize the gut with the left hand, and, keeping it on the stretch, separate it from its attachments by repeated incisions until we reach the duodenum, where the intestine is again cut off. The entire length of the intestines is then laid open with the enterotome along their attached border, the mucous membrane is cleansed with a stream of water and then examined.

The ordinary lesions are catarrhal, croupous, and ulcerative inflammation, perforations, strictures, new growths, and waxy degeneration. Caution is necessary in distinguishing normal from abnormal congestion. A very considerable amount of congestion may exist without disease. In cholera seasons, especially, observers are prone to call the most moderate degrees of congestion abnormal.

For a more superficial examination, the intestines need not be removed. We can slit up the lower end of the ileum and the colon and rectum, and examine them in place.

The intestines can be preserved for minute examination by cutting them into pieces a few inches long and suspending them in a solution of Potass. Bichrom. (gr. xv. to the ℥ i) and alcohol, equal parts.

The Stomach and Duodenum.—We now introduce the enterotome into the duodenum in its transverse portion, and open it on its convex border. When we reach the pylorus, the incision

is carried on along the greater curvature of the stomach as far as the œsophagus.

(If poisoning is suspected, a ligature should be placed on the lower end of the œsophagus, and at the middle of the small intestines, and these organs removed unopened. They are then to be opened in a clean glass jar, and after inspection of the mucous membrane, both stomach and contents are to be delivered to the chemist for examination.)

We now look for the orifice of the bile-duct, which will be found about the middle of the descending portion of the duodenum near the lower part of its concave border. Pressure on the gall bladder will cause the bile to flow into the intestine, if the ducts are pervious. A long director is then passed into the bile-duct and up into its hepatic branch, and the duct laid open. It is said that if the upper part of the duct is stained by the bile, while its duodenal extremity is not stained, this may indicate the existence of an inflammatory swelling of the duodenal portion during life sufficient to obstruct the flow of bile into the intestine.

The mucous membrane of the duodenum and stomach are now washed off and examined. Acute inflammation from caustic poisons, chronic catarrhal inflammation, hemorrhage, ulcers, and new growths are the lesions most frequently met with. In hospital patients, we sometimes find a diffused congestion of the stomach exactly like that produced by an irritant poison, as a result of small doses of croton-oil given just before death.

The stomach can be preserved entire if hung in a large jar in a mixture of equal parts of alcohol and Sol. Potass. Bichrom.

The Liver.—To remove the liver, we first divide the diaphragm on either side as far back as the spine. The liver is then drawn downward and the broad ligament and the right and left lateral ligaments divided. It is then turned backward into the thoracic cavity, the stomach and duodenum are seized and dissected off, the vessels on its under surface divided. The liver can then be lifted up, the coronary ligament and remaining vessels dissected away, and the organ removed.

To determine the actual size of the organ, it should be both measured and weighed. The size of the liver varies greatly in

different healthy individuals, but usually bears a relation to the size of the body. According to Frerichs, the relative weight of the liver to that of the body, in healthy adults, varies from one twenty-fourth to one fortieth. The absolute weight is from 1.8 to 4.6 lbs. avoird. In children, the relative weight of the liver is greater. The table on page 28 is taken from Frerichs.

The liver is increased in size and weight during the second stage of digestion; it is diminished in starvation, and is increased by congestion from any cause.

The surface of the liver is now examined, then the organ is laid on its lower surface and several long and deep incisions made transversely from the convex surface downward. After examining the liver tissue, the gall bladder is opened, and the character of the bile and the presence of biliary calculi noticed.

We often find the surface of the liver of a greenish or blackish color. Less frequently this abnormal color extends into the substance of the organ. This color is produced by the action of sulphuretted hydrogen on the coloring matter of the blood. In some cases we find granules of black pigment, especially along the course of the blood-vessels. These granules are composed of sulphate of iron. They are produced by the combination of sulphuretted hydrogen with the iron of the blood. Their character can be demonstrated by treating them first with hydrochloric acid and then with a solution of ferrocyanide of potash. After the addition of the latter solution, a blue color is produced. This entire process is a post-mortem one.

The Pancreas should now be examined. This organ is of oblong shape, flattened from before backward, its right extremity or head is broad, its left extremity or tail is narrow, the intermediate portions or body tapers between the two. Its length is from 6 to 8 inches, its breadth $1\frac{1}{2}$ inches, and its thickness from $\frac{1}{2}$ to 1 inch. Its weight is from 2 to 6 ounces. The organ may be rounded instead of flattened, the head and tail may be disproportionately large. The tail may be prolonged or may divide into several processes, or be curved. At the position of the mesenteric artery and vein, the pancreas is

TABLE I.

RELATIVE AND ABSOLUTE WEIGHT OF THE LIVER.*

AGE.	Weight of		Relative Weight of Liver to that of Body.	Weight of Spleen.	Relative Weight of Spleen to that of	Dimensions of Liver.				Dimensions of Spleen.					
	Body.	Liver.				Right Lobe.	Left Lobe.	Length.		Breadth.		Greatest Thickness.	Length.	Breadth.	Thickness.
								Right Lobe.	Left Lobe.	Right Lobe.	Left Lobe.				
5 months fetus	Kilog. 0.72	Kilog. 0.035	1:20.5	0.0025	1:288.0	1:14	2½	2	2	1½	5-6	7-12	Par. In. 5-6	Par. In. 7-12	Par. In. 5-12
6 "	" 1.3	" 0.060	1:21.6	0.004	1:325.0	1:15	3	2	2	2½	11-12	11-12	1 1-6	1	5-12
7 "	" 2.2	" 0.13	1:17.0	0.006	1:366.6	1:21.6	8	2½	2	1½	1	1	1	1	5-12
New-born child.	1.6	0.056	1:28.57	0.008	1:200.0	1:7	2½	2	2	1½	1½	1½	1½	1½	5-12
" " "	1.4	0.058	1:24.1	0.011	1:145.54	1:5.27	2½	2½	2½	2	1½	1½	1½	1½	5-12
1 day old.	3.8	0.185	1:20.5	0.011	1:345.4	1:16.8	2½	2½	2½	1½	1½	1½	1½	1½	5-12
8 days old.	2.7	0.103	1:26.1	0.009	1:248.6	1:11	2½	2½	2½	1½	1½	1½	1½	1½	5-12
5 weeks old.	1.95	0.090	1:21.66	0.016	1:121.8	1:5.62	2	2	2	1½	2½	2½	2½	2½	1
15 months old.	8.8	0.25	1:33.2	0.020	1:415.0	1:12.5	3½	3½	3½	2½	3½	3½	3½	3½	1
5 years old.	8.8	0.48	1:18.3	0.1	1:88.0	1:4.8	3½	4¾	4¾	1½	2½	2½	2½	2½	1
11 "	24.8	0.97	1:25.56	0.14	1:177.14	1:6.9	6½	6½	6½	3½	4½	4½	4½	4½	¾
22 "	64.5	1.6	1:40.3	0.16	1:140.3	1:10	...	...	...	...	...	...	...	...	¾
27 "	60.0	1.9	1:36.5	0.22	1:227.	1:8.6	...	...	...	...	...	...	...	...	¾
35 "	32.0	0.82	1:39.0	0.08	1:400.	1:10.2	6	5½	5½	2½	3½	3½	3½	3½	¾
36 "	55.5	1.5	1:37.0	0.15	1:370.	1:10	7	5½	5½	4	5	5	5	5	¾
44 "	56.2	1.4	1:40.1	0.25	1:324.8	1:5.6	7½	6	6	3½	3	3	3	3	¾
49 "	59.5	1.47	1:40.1	0.15	1:379.	1:9.8	7½	4¾	4¾	5½	5½	5½	5½	5½	¾
63 "	45.5	1.4	1:32.5	0.12	1:390.	1:11.6	7½	6	6	3	3	3	3	3	¾
80 "	30.1	0.7	1:43.5	0.1	1:305.	1:7	...	...	...	...	...	...	...	...	¾

* To reduce kilogrammes to pounds avoirdupois, multiply the numbers in the table by 2.2046.

To reduce Paris inches to English inches, divide the numbers in the table by .9375.

sometimes divided so that a portion of it lies behind these vessels. The head of the pancreas is embraced by and adherent to the concavity of the duodenum. The pancreatic duct usually joins the common bile-duct and has a common opening with it into the duodenum; sometimes, however, the two ducts have separate openings.

THE GENITO-URINARY ORGANS.

(1.) *The Male Organs.*—If the urine is to be examined, it should be drawn off with a catheter.

In many cases, it is sufficient to open the bladder on its upper surface, and examine it in place. If a more thorough examination is necessary, the bladder should be removed with the urethra. For this purpose, the penis may be removed entire, or the skin is dissected off from it nearly up to the glans penis, the penis is cut through at this point, and drawn out like a finger from a glove, leaving the skin and glans penis behind. That portion of the urethra which passes beneath the arch of the pubis may be carefully dissected away from the bones, or the bones may be sawn through on either side and the pubic arch removed, with the urethra, or the symphysis may be cut or sawn through and the pelvic bones violently separated. In this way, the urethra, prostate, vesiculæ seminales, and bladder are removed together.

The bladder and urethra should now be laid on the table, a long director passed into the urethra, the entire length of the canal laid open on its upper surface, and the incision prolonged into the wall of the bladder.

(2.) *The Female Organs.*—After observing the position of the uterus and ovaries, these are to be removed together. The uterus and bladder are seized and the soft parts dissected away from them so that the bladder, uterus, ovaries, ligaments, upper part of the vagina and rectum are removed together. The bladder is first laid open, then the rectum, then the vagina and uterus on their anterior surface, then the ovaries and Fallopian tubes. In puerperal cases, incisions should be made through the broad ligaments and at many points in the wall of the uterus, to ascertain the condition of the uterine sinuses.

The Uterus.—In the new-born infant, the uterus is small, the body flattened, the cervix disproportionately large. During childhood, the organ increases in size, but the body remains small in proportion to the cervix. At puberty, the shape changes, and the body becomes larger. Its length is then 2–3 inches, its breadth 16–20 lines, its thickness 8–12 lines. At every menstruation, the uterus is somewhat swollen and congested. After pregnancy, it does not return to its original size, but remains somewhat larger. In old age, it becomes smaller, its walls more fibrous.

The uterus is held in position by the broad and round ligaments, by its attachments to the bladder in front, the rectum behind, and the vagina below. The anterior lip of the cervix reaches to $\frac{3}{4}$ inch above the horizontal plane of the most superior point of the symphysis pubis. The long axis of the uterus is rather nearer to the posterior than to the anterior wall.

The shape of the uterus is that of a pear, flattened antero-posteriorly. The body is inclined forward at an angle of 140° with the cervix, while the latter is inclined somewhat backward.

The uterus is covered by the peritoneum anteriorly, to a point a little below the level of the os internum, posteriorly as far as the insertion of the vagina into the cervix. The peritoneum is firmly attached to the anterior and posterior surfaces of the uterus by a layer of connective tissue; at the sides, it separates to form the broad ligaments.

The greater part of the body and neck is made up of smooth muscular fibres, closely joined to each other. During pregnancy, these fibres are greatly increased in size. With the fibres we find connective tissue, with many cells and a few elastic fibres.

The mucous membrane of the body is composed of tubular follicles, either single or branching, lined with cylindrical epithelium, which in some animals is ciliated. The internal surface of the body and neck is covered with a layer of ciliated epithelium, which, near the external os, changes into pavement epithelium.

The mucous membrane of the neck is thrown into folds, in which are contained mucous follicles, simple tubes lined with

cuboidal epithelium. In the lower half of the cervix there are small papillæ covered with ciliated epithelium, between the openings of the mucous follicles. We also find in the mucous membrane closed, rounded follicles, (ovula Nabothi,) containing mucus and epithelium, which are regarded as retention cysts of the mucous follicles, or as new growths. The outer surface of the cervix contains no glands, but is covered with small papillæ. At every menstruation the mucous membrane undergoes a rapid growth, followed by a rapid degeneration. The changes produced in the uterus by pregnancy are so fully described in works on obstetrics that it is unnecessary to speak of them here.

The Ovaries are two flattened oval bodies, which are placed one on each side, nearly horizontally, at the back of the broad ligament of the uterus, and are enveloped by its posterior membranous layer. They are largest in the virgin state; their weight is from 3-5 scruples, and they measure about $1\frac{1}{2}$ inches in length, $\frac{3}{4}$ inch in breadth, and nearly $\frac{1}{2}$ inch in thickness. Each ovary is free on its two sides, and also along its posterior border; but it is attached by its anterior border. Its inner end is attached to the ligaments of the ovary. To its outer extremity is attached one of the fimbriæ of the Fallopian tube.

The ovary is covered by the peritoneum, except at its attached border. Beneath this is a dense fibrous capsule. The ovary proper consists of a connective tissue stroma, in which, according to some writers, are smooth muscular fibres. Scattered through the stroma are the Graafian vesicles containing the ova.

In adult females, we usually find corpora lutea in their various stages.

The Fallopian Tubes are between three and four inches in length. They commence at the upper angles of the uterus as small cords, which become larger, bend backward and downward toward the ovary. They terminate in an expanded fimbriated extremity, about an inch beyond the ovary.

The tubes are covered by the peritoneum. Their walls are composed of connective tissue and smooth muscular fibres. Their inner surface is thrown into folds, and lined

with ciliated epithelium, continuous with the uterine mucous membrane.

THE EXAMINATION OF THE BODIES OF NEW-BORN INFANTS.

In examining the bodies of new-born children, we have to consider, besides the ordinary lesions of disease, the age of the child; whether it was born alive; how long it has been dead; what was the cause of death.

EXTERNAL INSPECTION.

The Size and Age.—Casper gives the following description of the foetus during the different months of intra-uterine life:

At the fourth week, the embryo is 4–6 lines long. The cleft of the mouth and two points representing the eyes can be recognized in the head. The extremities are represented by little wart-like projections. The heart can be distinguished; the liver is disproportionately large. The umbilical vessels are not formed. The entire ovum has the size of a walnut.

At the eighth week, the embryo is 15–18 lines long. The head forms more than a third of the entire body. The nose and lips can be distinguished, but not the external ear. The hand is longer than the fore-arm, the fingers are formed, but joined together by a gelatinous mass; the toes look like little buds; the soles of the feet are turned inward. The arms appear as points. The abdomen is closed. All the viscera can be recognized. Points of ossification are formed in the apophysis of the first cervical vertebra, the humerus, radius, scapula, ribs, and cranial bones. There are rudimentary external genitals, but the sex can hardly be distinguished. The ovum reaches the size of a hen's egg.

At the twelfth week, the placenta is formed. The embryo is 2–2½ inches long, and weighs three ounces. The eyes and mouth are closed. The neck separates the head from the thorax. The nails can be recognized on the fingers. The sex can be recognized. The umbilical cord is inserted near the pubes. The

thymus and the suprarenal capsules are formed. The cerebellum, cerebrum, medulla, and the cavities of the heart can be recognized. The humerus is $3\frac{1}{2}$ lines long; the radius, $2\frac{1}{2}$ lines; the ulna, 3 lines; the femur, 2–3 lines; the tibia, 2–3 lines; the fibula, $2\frac{1}{2}$ lines. The ovum is as large as a goose's egg.

At the sixteenth week, the embryo is 5–6 inches long, and weighs $7\frac{1}{2}$ –9 ounces. The skin is of a rose-red color; there is a commencement of formation of fat in the subcutaneous tissue. The scrotum and labia are formed. The face begins to assume its proper appearance. There is meconium of a white color in the duodenum. The liver is not so disproportionately large; the gall bladder is formed; the anus is open. The length of the humerus is 8 lines; of the radius, 8 lines; of the femur, 4–5 lines; of the tibia, 4–5 lines. The calcaneus begins to ossify at the middle of the fourth month.

At the twentieth week, the embryo is 10–11 inches long; it weighs from 21–30 ounces. The nails are very perceptible. There is a thin down on the head. There is not yet any of the vernix caseosa. The secretion of bile has commenced, and stains the meconium. The insertion of the umbilical cord is further off from the pubes. The convolutions of the brain cannot be recognized. The humerus is 13–15 lines in length; the radius, 12 lines; the ulna, 13 lines; the femur, 12 lines; the tibia and fibula, each 12 lines. The astragalus begins to ossify; also the upper part of the sternum.

At the twenty-fourth week, the embryo is 12–13 inches long, and weighs $1\frac{1}{2}$ – $1\frac{3}{4}$ pounds. The lanugo and the vernix caseosa are formed. The skin is of a dusky cinnabar red color. The meconium is darker. The scrotum is empty, small, and red; the labia majora are prominent. The pupillary membrane is present and easily recognized. The length of the humerus is 16 lines; of the radius 16 lines; of the ulna, the femur, the tibia, and the fibula, each 17 lines.

At the twenty-eighth week, the embryo is 14–15 inches long, and weighs 3– $3\frac{1}{2}$ pounds. The hair is more abundant and longer. The large intestine is filled with meconium. The humerus is 20–22 lines long; the radius, 17 lines; the ulna, 18 lines; the femur, tibia, and fibula, each 19–21 lines.

At the thirty-second week, the embryo is 15-16 inches long, and weighs 3-5 pounds. The skin is of a lighter color. The pupillary membrane has disappeared. The testicles are in the scrotum, or the inguinal canal. The nails nearly reach the ends of the fingers. The humerus is 23-24 lines long; the radius, 18-19 lines; the ulna, 22-23 lines; the femur, 24 lines; the tibia and fibula, each 21-23 lines. The last sacral vertebra begins to ossify.

At the thirty-sixth week, the embryo is 17-18 inches long, and weighs about 6 pounds. The scrotum begins to become wrinkled, and the labia to close. The hair becomes longer, while the lanugo begins to diminish in amount.

At the fortieth week, the fœtus is fully developed, and the term of its intra-uterine life accomplished.

The fresh corpse of a new-born child at term no longer resembles that of the immature fœtus. The skin is firm and pale, like that of an adult. The lanugo has disappeared, except on the shoulders. The average length is 19 1-7 inches; the weight, 7 pounds. The nails are hard, and reach to the points of the fingers, but not to those of the toes. The cartilages of the ears and nose are hard. The point of ossification in the lower epiphysis of the femur should be noticed. If it is absent, the fœtus is, as a rule, not more than thirty-seven weeks old; but in rare cases, it may be absent at term. A point of ossification $\frac{1}{2}$ line in diameter indicates an age of 37-38 weeks, if the child was born dead. Rarely it is no larger than this at term. A diameter of $\frac{3}{4}$ -4 lines indicates an age of 40 weeks at birth. A diameter of more than 4 lines indicates, as a rule, that the child has lived some time after its birth. The pupillary membrane has disappeared. The testicles are in the scrotum. The labia cover the clitoris.

Twenty-four hours after the birth of the child, the skin is firmer and paler. The umbilical cord is somewhat shrivelled, although still soft and bluish-colored.

From the second to the third day, the skin has a yellowish tinge, and the cuticle sometimes appears cracked. The umbilical cord is brown and dry.

From the third to the fourth day, the skin is more yellow, and

the cuticle separates from the skin and abdomen. The umbilical cord is of a brownish red color, flattened, semi-transparent, and twisted. The skin around its insertion is red and congested.

Further Inspection of the Body.—The head should be examined for the marks of injuries. Very commonly some portion of the scalp will be found swollen and infiltrated with blood and serum. This is the caput succedaneum formed during delivery. The mouth and nose should be examined for the presence of any foreign bodies which might have caused suffocation.

The neck should be examined for the marks of strangulation. The umbilical cord may be twisted around the child's neck and strangle it. The mark left by the umbilical cord is usually continuous, broad, soft, not excoriated, sometimes accompanied by ecchymoses in the skin.

The entire body should be examined as to the presence of vernix caseosa, blood, marks of injuries, and the existence of putrefaction.

The umbilical cord may be found cut or torn. It usually separates by the fifth day, sometimes not until the tenth. If the umbilicus is cicatrized and healed, the child has probably lived for three weeks. A zone of redness around the insertion of the cord may exist previous to birth. Redness with swelling and suppuration can only be found in a child which has lived for several days. The drying and mummification of the cord may take place as well in dead as in living children. It is possible for a child to die by hæmorrhage from a cut or torn cord either before or after it has breathed.

The extremities may exhibit fractures of their bones. This may take place during intra-uterine life, from injuries to the woman, or from unknown causes; or may be produced by a violent delivery, or by injuries after birth.

INTERNAL EXAMINATION.

The Head.—The fontanelles and sutures should first be examined as to their size and for penetrating wounds. An incision should then be made through the scalp across the vertex, and the flaps turned backward and forward. With a small knife the

edges of the bones should be separated from the membranous suture and the dura mater, beginning low down in the frontal suture and going back into the lambdoidal suture on either side. The bones are then drawn outward and cut through with a pair of strong scissors. The brain is removed as in the adult.

Effusions of blood—cephalhæmatoma—may be formed soon after birth between the pericranium and bone, or more rarely between the dura mater and bone. Clots are also found between the dura mater and skull, between the dura mater and pia mater, and more rarely in the substance of the brain as the result of protracted or instrumental deliveries, or of injuries after birth.

The cranial bones may be malformed, or exhibit the lesions of ricketts or caries, or be indented, fissured, or fractured. The indentations, fissures, and fractures may be produced during intra-uterine life by injuries to the mother, or by unknown causes; by difficult deliveries, and by direct violence after birth.

The brain is naturally much softer and pinker than in the adult; it may be much congested or anæmic without known cause. The ventricles contain very little serum. Malformations, apoplexies, hydrocephalus, and inflammatory lesions are to be looked for.

The pharynx should be opened and examined for foreign bodies.

The thyroid gland weighs about 3 iij. It may be enlarged so as to interfere with respiration.

The larynx and trachea should be examined for the lesions of inflammation and for injuries to the cartilages.

The Thorax should be opened as in the adult.

The thymus gland occupies the upper portion of the anterior mediastinum, covering the trachea and large vessels. Its average weight is half an ounce. It may be hypertrophied and compress the large vessels, or be inflamed and suppurating.

The heart lies more in the median line than in the adult. It weighs $1\frac{1}{2}$ – $3\frac{1}{2}$ ounces. The ventricular walls are of nearly equal thickness. The pericardium contains very little serum. The serum may be increased in amount and stained red by decom-

position. There may be small extravasations of blood beneath the pericardium in still-born children, and in those born alive. Pericarditis with effusion of serum and fibrine may exist before birth.

The time of the closure of the foramen ovale and of the ductus arteriosus varies very widely in different cases.

Endocarditis with consequent changes in the valves may exist during intra-uterine life. Malformations and malpositions of the heart-cavities and large vessels are not infrequent.

The pleural cavities contain very little serum. Decomposition may increase the amount of serum and stain it red. Small extravasations of blood in the sub-pleural tissue are found in children which have died before birth, and after protracted labors. Inflammation with exudation of serum, fibrine, and pus may exist before birth.

The Lungs in a still-born foetus are small, do not cover the heart, are situated in the upper and posterior portion of the thorax, are of a dark red color and of firm, liver-like consistence, and do not crepitate. In a child born alive and which has respired freely, the lungs fill the thoracic cavity, are of a light red color, and crepitate. If respiration has been incompletely performed, we find various intermediate conditions between the foetal and the inflated states.

If there is any doubt as to respiration having taken place, it is customary to employ the hydrostatic test. This is done by placing the lungs, first together and afterward cut into small pieces, in water. If they sink, the child has not breathed; if they float, it has. The test is not, however, a certain one. Taylor says:

1. That the hydrostatic test can only show whether a child has or has not breathed, not whether it was born alive or dead.

2. That the lungs of children who have lived after birth may sink in water, owing to their not having received air, or to their being in a diseased condition.

3. That a child may live for some time with the lungs only partly inflated.

4. That a child may live for twenty-four hours, when no part of its lungs has been penetrated by air.

5. The sinking of the lungs is no proof that a child has been born dead.

6. That the lungs of children which have not breathed, and have been born dead, may float in water from putrefaction or artificial inflation.

The lesions of inflammation, and vesicular and subpleural emphysema may be found in the lungs of new-born children.

The Diaphragm.—In still-born infants the convexity of the diaphragm reaches the fourth or fifth rib. After respiration, it reaches a point between the fourth and seventh rib. Its position is so variable that it is of little diagnostic value.

The Peritoneum contains a very little clear serum. Bloody serum may be produced by decomposition. The peritoneum is often the seat of intra-uterine inflammation.

The Stomach.—The mucous membrane of the stomach may be intensely congested as a natural condition. In still-born children it contains a little mucus, sometimes meconium. The contents of the stomach should be examined microscopically in doubtful cases, for the presence of milk and starch. Inflammation and swelling of the closed follicles of the mucous membrane sometimes occur.

In the small intestine, inflammation and swelling of the solitary and agminated follicles are sometimes found.

The large intestine usually contains meconium, but this may be evacuated before or during birth.

The formation of gas in the stomach and intestines does not usually take place until respiration is established. If decomposition has commenced, however, gas may be formed as a part of the process.

The Liver is of a dark red color, full of blood, and large. Its size diminishes after respiration is established. The size is so variable before and after respiration, that it gives little information as to the age of the child. Large extravasations of blood are sometimes found beneath the capsule of the liver without known cause. A variety of pathological conditions, fatty and waxy degeneration, gummy tumors, etc., may be found.

The Spleen is large and firm. It may be abnormally large, and its capsule is sometimes covered with fresh exudation.

The Suprarenal Capsules are large. They may be dilated into large cysts filled with blood.

The Kidneys are lobulated. There may be ecchymoses on their surfaces; inflammation and suppuration; deposits of uric acid and urates in the tubes of the pyramids; cystic dilatation of the tubes, sometimes reaching an enormous size.

The Bladder may be full or empty, both in still-born children and in those which have breathed. Dilatation and hypertrophy may exist during intra-uterine life.

Spinal Cord.—Extravasations of blood between the membranes of the cord may occur from the same causes as in the brain. Spina Bifida is the most frequent malformation.

PART II.

MORBID ANATOMY OF THE ORGANS.

THE NERVOUS SYSTEM.

THE MEMBRANES OF THE BRAIN.

THE DURA MATER.

WHEN the Calvarium is removed, the Dura Mater is brought into view. It will be found somewhat adherent to the bone; sometimes very adherent, as a senile change, or as the result of chronic inflammation. At the vertex the Dura Mater is often thinned or even perforated by the pacclyonian bodies, which may also produce indentations in the internal surface of the calvarium.

Hæmorrhages.—These occur in the form of clots between the Dura Mater and skull, or, more rarely, in the substance of the membrane. They are usually produced by direct violence applied to the skull, and correspond in situation to the external injury. In children, however, clots are found between the dura mater and parietal bones, in connection with cephalhæmatoma of the pericranium. Severe injuries and penetrating wounds of the skull may also lacerate or penetrate the dura mater. In rare cases the membrane has been found ruptured by the pressure of fluid in hydrocephalus.

Thrombosis of the sinuses of the dura mater is not uncommon. The thrombi are yellow and firm, or soft, broken down, and puriform. They are produced by changes in the blood, by pulmonary disease, by inflammation of the skin of the face and scalp, with thrombosis of the veins, by inflammation and fracture of the cranial bones, by meningitis, by otitis, and occur without known cause.

The thrombi may cause meningitis, small apoplexies, encephalitis, and multiple pyæmic infarctions. Or a thrombus may be found in the vein after death, which has apparently produced no lesions.

I have seen a case in which a young female domestic, without any known cause, suddenly became unconscious, comatose, her right arm and leg paralyzed; her pulse rose to 140, her temperature to $103\frac{1}{2}$ F., and she died on the ninth day. At the autopsy the straight sinus of the tentorium was found filled with a large, white thrombus, which extended through all the branches given off from the corpora striata and optic thalami. The upper portion of both corpora striata, of the left optic thalamus, and of the fornix, were in the condition of red softening. There were no other lesions throughout the entire body.

Inflammation.—Pachymeningitis may involve either the external or internal layers of the Dura Mater. Inflammation of the external layers is secondary to injuries or diseases of the cranial bones. Fractures of the skull, either depressed or not, osteitis, caries, chronic otitis, and even external periostitis may produce it. The dura mater is congested, thickened, softened, and ecchymotic. The inflammation may go on to form pus between the membrane and the bone and in the substance of the membrane, sometimes accompanied with thrombosis of the neighboring venous sinuses, sometimes with gangrene of the dura mater. Such inflammations may extend to the pia mater and brain, or, the inflammation may merely produce fibrous thickening, with or without pigmentation of the Dura Mater and adhesions to the skull.

Inflammation of the internal layers of the Dura Mater occurs as a complication of pyæmia, puerperal fever, chronic nephritis, and the exanthemata. The inner surface of the membrane appears streaked with delicate vessels, is of a rosy color, and is lined with a layer of exudation, which is either delicate, grayish, and soft, like mucus, or more consistent and membranous, or yellow, loose, and puriform.

There is also a primary form of inflammation of the internal layer of the dura mater, called Pachymeningitis chronica hæmorrhagica. In this form there is found, loosely adherent to

the internal surface of the dura mater, a thin membrane, grayish with red dots, looking like coagulated fibrine. This is composed of a homogeneous basement substance containing connective tissue-cells and a great number of capillary vessels, with numerous small hæmorrhages. In a more advanced stage this new membrane may become enormously thickened, its outer layers change into fibrous tissue, and the hæmorrhages in its substance may attain such a size as seriously to compress the brain, and even cause death. Very rarely serum accumulates between the layers of new membranes, and in this way cysts of large size can be formed. In rare cases, diffuse suppuration of the entire new membrane has been observed. The slighter degrees of this form of inflammation may occasion no symptoms during life. The more advanced lesions usually occur in idiots, epileptics, etc.

NEW GROWTHS.

Cysts of the Dura Mater have only been described in a few rare instances.

Fibromata, also, are very seldom met with.

Osteoma.—New formation of bone may occur either in the inner or outer layers of the Dura Mater. In the inner layers it occurs in the form of plates, or, more rarely, globular tumors, most frequently in the falx, but also in other situations. These plates usually cause no symptoms ~~during life~~.

Bony growths in the outer layers are of irregular shape, and often become continuous with the neighboring bones.

Lipomata have been observed in the form of small globular tumors attached to the inner surface of the dura mater.

Syphilitic Growths (Gummata) are of very various sizes and shapes. They are usually firm, partly of a gray semi-transparent color, partly yellow and cheesy. They grow either from the outer layers of the Dura Mater, and may then cause thinning, erosion, osteo-myelitis, caries, and necrosis of the bones; or from the inner layers, and invade the brain, causing secondary tumors and inflammation of the pia mater and brain.

Psammoma.—Under this name Virchow has described a form

of tumor growing inward from the Dura Mater. These tumors are small, of irregular globular shape, of firm, gritty consistence, of white or pinkish color. They are formed of a loose fibrous tissue, of round and fusiform cells, and of globular calcareous concretions, such as are found scattered through the normal brain tissues. These bodies are arranged at the ends of broad fibres, as if on stalks.

Sarcoma.—These tumors grow from the inner surface of the dura mater, invading the brain, or from its outer surface, thinning or perforating the skull. They are very hard and white, or soft and pinkish, or partly cheesy. They are composed of round, oval, or fusiform cells, sometimes of myélopaxes, mixed with a variable amount of fibrous tissue.

Carcinoma.—This form of new growth may occur as a primary or secondary new growth in any part of the Dura Mater. The tumors may grow inward and compress the brain, or outward and project through the skull as a fungous, bleeding mass. Less frequently the membrane becomes infiltrated with the new growth forming large flat tumors, which may invade the bone. They are either hard, or soft and vascular. They usually consist of nucleated cells arranged in alveoli, but sometimes have the structure of epithelial cancer.

THE PIA MATER.*

When the Dura Mater is stripped backward, it will be found somewhat adherent along the longitudinal fissure. The Pia Mater is then brought into view. This membrane will be found in various degrees of congestion. The vessels may be nearly empty or distended with blood. Generally the degree of post-mortem congestion is not a certain evidence of the amount of congestion during life. Along the edges of the longitudinal fissure, and more rarely on the under surface of the brain, are

* I have followed the plan, now generally adopted, of not considering the arachnoid as a serous sac with visceral and parietal layers, but of treating of only two membranes, the dura and pia mater. The dura mater has an epithelial lining on its internal surface, the pia mater has two layers, a membranous and a vascular.

a number of small, white, firm, irregular bodies—the pacchyonian bodies. They may be few and small, or numerous and large, and scattered over the convex surface of the hemispheres. They may perforate the Dura Mater, or, more rarely, the wall of the longitudinal sinus. They are composed of fibrous tissue, but may undergo fatty, or calcific degeneration. The pia mater is also frequently thickened, opaque and white, especially along the course of the vessels. The pacchyonian bodies and the opacities of the Pia Mater are both said to be dependent upon repeated congestions of the membrane. They are most frequently found as senile changes, and do not indicate the pre-existence of any disease. The amount of serum beneath the pia mater and infiltrating its tissue is very variable in amount. It is not infrequent to find in hospital patients suffering from chronic nephritis, cardiac, or pulmonary disease, or chronic alcoholism, a very considerable amount of serum in this situation, and yet the patient has been free from cerebral symptoms. In other cases again this same serous effusion affords the only explanation of grave cerebral symptoms. It is necessary to be very careful in judging of the importance of this accumulation of fluid.

Hæmorrhage.—Most authors speak of hæmorrhages between the dura and pia mater as of rare occurrence, except from violence or the rupture of small aneurisms in the pia mater. This opinion appears to me to be erroneous; and I have found clots in this situation not infrequently without any special cause to account for them.

Hæmorrhages in the substance of the pia mater occur spontaneously in young children, but in adults are usually the result of violence.

Hæmorrhages between the pia mater and brain are produced by violence, by apoplexies in the brain substance, or occur by themselves. In the latter case, the clot is usually situated at the base of the brain and around the medulla. Some of the blood may escape outside of the pia mater between this membrane and the dura mater.

Meningitis.—Inflammation of the pia mater causes the pro-

duction of exudation in the substance of and beneath the pia mater. This exudation is composed of serum, lymphoid cells, and coagulated fibrine in variable proportions. It occupies circumscribed portions of the pia mater, or is distributed along the course of the vessels, or extends over a large part of the brain and upper part of the spinal cord. It gives to the pia mater a gray, yellow, or greenish color. It is rare to find any exudation on the free surface of the membrane. The inflammation may involve the superficial layers of the brain. This disease usually occurs in adults. It is either idiopathic or secondary to inflammations of the dura mater and cranial bones, to thrombosis of the large venous sinuses, to severe concussions of the head, or occurs as epidemic cerebro-spinal meningitis.

This inflammation is usually fatal. In rare cases, collections of pus form between the pia mater and brain, and exist as abscesses for a considerable time. Sometimes the exudation undergoes cheesy degeneration, and forms firm yellow masses in the sulci. Sometimes the inflammation subsides entirely, leaving the pia mater thick, opaque, and adherent to the brain and dura mater.

Chronic Meningitis.—This is a disease of which our knowledge is imperfect. It is not always easy to draw the line between the lesions of chronic inflammation and those of a more degenerative character.

The most marked form of chronic meningitis is seen in the general paralysis of the insane. Here we find the pia mater thick, opaque, and adherent to the brain. The outer layers of the brain are softened or sclerosed; and there are peculiar changes connected with the small blood-vessels.

Another form of chronic meningitis occurs by itself. It is most frequent in old people. The patient is usually sick for some time, but the diagnosis is often not established during life. The pia mater is thick, opaque, and adherent to the brain. There is a moderate amount of turbid serum beneath the pia mater, and sometimes small collections of pus. The pia mater is infiltrated with pus cells. I have seen in some cases dilatation of the vessels and increase of cells in the superficial layers of the brain.

Tubercular Meningitis.—This form of meningitis accompanies the production of gray miliary tubercles in the pia mater. The tubercles are found most abundant at the base and in the sulci, and are sometimes hardly to be seen with the naked eye. They are very small transparent granulations surrounding the small blood-vessels. They are formed by an accumulation of small round cells within the perivascular sheaths. Accompanying the tubercles, serum, fibrine, and lymphoid cells form a turbid fluid exudation beneath and in the pia mater. This form of inflammation is most frequent in childhood, but occurs also in adult life. It is usually found with tuberculosis of the other viscera.

NEW GROWTHS.

Fibroma, Angioma, Sarcoma, Lipoma, Cysts, Carcinoma, and Epithelioma occur in the pia mater in rare cases. Gummy Tumors are found in and beneath the pia mater. Tumors formed of cholesterine and epithelial cells have been found at the base of the brain. Small plates of bone are sometimes formed in the pia mater over the convex surface of the brain.

Tubercular tumors occur as rounded bodies, apparently formed of an aggregation of smaller tumors. They are usually far advanced in cheesy degeneration so as to have an opaque yellow color. They are most frequent in children.

PARASITES.

The cysticercus cellulosæ has been observed in the pia mater.

THE VENTRICLES OF THE BRAIN.

THE EPENDYMA AND THE CHOROID PLEXUS.

The ventricles usually contain a small amount of clear serum. If there is inflammation of the pia mater, especially at the base of the brain, turbid or purulent serum is frequently found in the ventricles. A considerable amount of clear serum in the ventricles, and even a moderate dilatation of these cavities, are not necessarily evidences of disease.

Pathological Accumulations of Serum are :

(1.) *Congenital Hydrocephalus*.—The highest degrees of this form will be considered with the malformations. The lesser degrees commence as a moderate effusion of serum into the ventricles, which increases to a greater or less extent, and for a variable time after the child's birth. In these cases, we find all the ventricles of the brain dilated and filled with clear serum. The brain substance forms a thin shell around these sacs of fluid. The ependyma of the ventricles is either smooth or thickened.

(2.) The serum accumulates after birth, usually in badly-nourished children.

The effusion may take place rapidly, with febrile symptoms. The serum is in variable amount, and clear; the walls of the ventricles, and sometimes the brain, are œdematous and softened; the pia mater shows no evidence of inflammation. Such an effusion of serum may be fatal, or may be absorbed, or may take on a chronic character.

(3.) The effusion may be caused by acute or chronic hyperæmia of the brain and its membranes. It may occur at any age. It has been ascribed to excessive bodily and mental exertion, alcoholism, tumors of the brain, obstruction of the venous sinuses and jugular veins, and to heart and lung diseases. The accumulation of serum is usually gradual, though the patient may at the last die suddenly. Such cases have been called serous apoplexy.

Atrophy of the brain is usually accompanied by a serous effusion in the ventricles.

The accumulation of serum in all the cases mentioned occurs equally in both ventricles. In rare cases, however, the effusion is confined to one lateral ventricle, or to the third ventricle, or to one of the cornua of a lateral ventricle. The dilated portion of the ventricle may then look like a cyst with a narrow opening.

Hæmorrhages into the ventricles take place when apoplexies of the brain substance break through into these cavities.

Inflammation of the Choroid Plexus and Ependyma is usually secondary to a meningitis, but may be primary. The ventri-

cles contain a moderate amount of serum made turbid by pus globules and broken-down particles of tissue. The ependyma is coated and infiltrated with the same material. The layer of brain tissue next the ependyma is softened or stained with small apoplexis. Or the entire brain may be softened and œdematous. Sometimes the ependyma is coated with a thick layer of lymphoid cells and fibrine. I have seen, in an adult, the ependyma thickened, of a mottled gray and red color; the brain substance next it soft and red; very little serum in the ventricles; no lesion in any other part of the brain; and the patient died comatose.

NEW GROWTHS.

Connective Tissue may occur as a new growth, causing a uniform thickening of the ependyma, or the thickening may be in plates, or in small granulations. In rare cases, small fibrous tumors are attached to the ependyma by narrow pedicles, and these tumors may become free. The new growth of connective tissue may cause adhesions between the walls of the ventricles. Calcification and ossification of the thickened ependyma has been seen in a few instances.

Lipoma.—Small, fatty tumors have been found attached to the ependyma and choroid plexus.

Enchondroma has been observed in the choroid plexus in small nodules.

Cystoid Growths occur very commonly in the choroid plexus in the form of little transparent vesicles. These little vesicles are not really cysts, but are composed of small, transparent cells closely packed together. These bodies have no pathological significance. A cyst, as large as a walnut, filled with colloid matter, and a dermoid cyst containing hair, have been observed in this situation.

Angioma may occur in the choroid plexus.

Carcinoma has been seen in very rare cases in the plexus of the fourth ventricle, and the ependyma of the lateral ventricles.

THE BRAIN.

MALFORMATIONS.

Cyclopia.—This malformation consists in an arrest of development affecting the cerebrum, which, instead of separating into two hemispheres, remains single, and the rudiments of the eyes become joined and form one eye. This single eye is in the middle of the face at the root of the nose, in a single orbit. Over this is an irregular body representing the nose. The rest of the face is well formed. Or the eye-ball may be wanting entirely, or there are two eyes joined together, or, more seldom, two separate eyes. The orbit is surrounded by rudiments of four eye-lids. The frontal bone is single, the nasal bones undeveloped, the ethmoid, vomer, and turbinated bones are absent. The optic nerve is double, single, or absent. There may be hydrocephalus. Such children are incapable of any prolonged existence.

Anencephalia.—This malformation may be of various degrees. The brain may be entirely absent, and the base of the cranium is covered with a thick membrane, into which the nerves pass. Or, the membranes may form a sort of cyst containing blood and serum, or portions of brain. Of the cranial bones, only those which form the base of the skull are present. The fœtus cannot live.

Hydrencephalocèle.—(1.) The cranial bones are incompletely developed. The cranium is flattened or concave from above downward. The brain is incomplete, or hydrocephalic, or replaced by a bloody, spongy mass, or by a sac of serum. It escapes from the cranial cavity by an opening in the cranium, usually lies on the neck, and may or may not be covered with skin. The fœtus is not viable.

(2.) The cranial bones are well developed, as is also the brain. At some point in the cranial bones, at the root of the nose, the occiput, on the sides of the skull, in the sagittal suture, or at the base, there is an opening through which escapes a sac composed of the membranes of the brain, inclosing serum or brain tissue. Or a portion of brain alone may protrude, covered by its membranes and the skin. These tumors may be small, or

as large as the child's head. The malformation is caused by a partial hydrocephalus of the cavities or membranes of the brain.

Hydrocephalus.—Hydrocephalus internus consists in an accumulation of serum in the ventricles of the brain. It occurs in all degrees, according to the period of foetal life at which it begins. There may be only a sac formed by the membranes, or the brain may be partly developed, or it may be perfect. The cranial bones are separated at their sutures and fontanelles. In rare cases, only part of one lateral ventricle is hydrocephalic, giving to the head a protuberance on one side. The viability of the foetus depends upon the degree of the hydrocephalus. Hydrocephalus externus is an accumulation of serum beneath the pia mater, or, according to some authors, between the pia and dura mater. It causes dilatation of the cranium and compression of the brain. It is of very rare occurrence.

Microcephalia.—This is an abnormally small size of the brain, with a correspondingly small cranium. The diminution in size affects principally the cerebral hemispheres, though the other parts of the brain are also small. The convolutions are few and simple, the cavities often dilated with serum, on the membranes are traces of inflammation. The cranium is small, the face large, the rest of the body small. The malformation is caused by inflammation or dropsy of the brain during foetal life. It is endemic in some countries, but single cases may occur anywhere. The foetus is viable.

Absence of portions of the brain may occur not only in idiots, but in persons whose minds are perfect.

HYPERTROPHY AND ATROPHY.

Hypertrophy.—In children, either before or after birth, less frequently in youth, and very seldom in adults, we find hypertrophy of the brain. This hypertrophy consists in an increase of size of the white substance of the hemispheres. If it takes place before the ossification of the cranium, the bones are separated at the sutures and fontanelles; if after this, the inner table of the skull is eroded and thinned. When the cranium is opened, the dura mater appears tense and anæmic, the convolutions of the brain are flattened, the brain substance is firm

and anæmic, the ventricles are small, the ganglia and cerebellum are either of normal size or compressed.

The disease is usually very chronic, and destroys life with symptoms of compression of the brain. There may, however, be acute exacerbations. The hypertrophy consists in an increase of the neuroglia.

Rokitansky has also described cases of hypertrophy as caused by tumors of the brain and compression of the medulla oblongata.

Hypertrophies of portions of the brain have been described by various authors.

Atrophy.—This may occur as a senile change, or in adults from idiocy, alcoholism, opium or lead poisoning, or chronic meningitis. In children who are very much reduced by chronic diseases, atrophy of the brain may accompany atrophy of the rest of the body.

The atrophy affects principally the cerebral hemispheres. The convolutions are small, the sulci broad, the ventricles dilated, the brain tissue firm, the gray substance discolored, the white substance dark colored, the blood-vessels dilated. The large ganglia at the base are also small. Serum usually accumulates beneath the pia mater and in the ventricles, the pia mater and often the skull become thickened; the brain substance may be œdematous, or contain hemorrhages.

INJURIES.

Wounds of the brain may be inflicted in a great variety of ways, and by many different instruments. The brain may be directly wounded by the foreign body, or indirectly by fragments of bone driven into it. It is very difficult to estimate the degree of injury which must cause death; persons frequently die from slight, and may recover from severe, penetrating wounds of the brain.

After wounds which remove a portion of the cranial bones, it is not uncommon, after a few days, to see a bleeding, fungus mass project through the opening. This mass, or hernia cerebri, consists of granulation tissue, brain matter, and blood. The brain tissue below it is softened and purulent; there is often an abscess in the brain substance beneath.

Severe injuries of the head which fracture the skull without displacing the fragments, or even cause no fracture, may produce laceration of the brain. The laceration takes place either at the point of injury or at some other part of the brain. We find small or large portions of the surface of the hemispheres mottled with little hæmorrhages, the brain tissue softened and broken down.

HÆMORRHAGE.

Effusions of blood in the substance of the brain are very common and may occur at any age. They may be of small size—capillary apoplexies, or consist of large blood-clots. They may occur in the large ganglia, or at any point in the hemispheres. They usually produce softening and breaking down of the brain tissue, and often find their way into the ventricles or beneath the pia mater. They cause death when of large size. The smaller ones sometimes undergo degenerative changes, the blood becomes absorbed, and there are left little cysts containing serum, surrounded by indurated, reddish-yellow brain tissue, or there are little spots of a reddish color composed of a soft, friable material.

These hæmorrhages are produced by fatty, or atheromatous degeneration of the vessels of the brain, hypertrophy, and valvular disease of the heart, convulsions, and external injuries. Often no satisfactory cause can be discovered.

INFLAMMATION.

Idiopathic inflammation of the brain is so rare that its existence is denied by some authors. The alterations in the brain tissue are usually secondary to injuries, to inflammation of the meninges, or to some obstruction of the cerebral vessels. The changes met with are, in recent cases, diminution of consistence and change in color; in chronic cases, increase of consistence.

Injuries, which either directly bruise and lacerate the brain, or do so indirectly without fracture of the skull, render the brain tissue soft, mottled with small apoplexies, the nervous tissue disintegrated and degenerated, the softened mass infiltrated with red blood globules and lymphoid cells.

Obstruction of the Cerebral Vessels occurs in a variety of

ways. The arteries may be occluded by emboli or thrombi, the capillaries by emboli, the veins and sinuses by thrombi. Or, the arteries and capillaries may be the seat of atheroma and fatty degeneration.

Emboli are usually portions of fibrine detached from the aorta or left heart, or from the pulmonary veins, or, rarely, in carcinoma of the lungs, portions of the new growth may be swept by the current of blood from the pulmonary veins. Sometimes, however, we can find nothing in the heart or vessels to account for the origin of the embolus.

Thrombosis of the arteries is produced by atheroma of their walls, by dilatation or fatty degeneration of the heart, and by certain conditions of the blood occurring in ill-nourished and cachectic persons. All these conditions are most likely to occur in old age.

Thrombosis of the veins and sinuses is caused by inflammation of the dura mater, cranial bones, and pericranium, or by unknown changes in the quality of the blood.

Fatty and calcific degeneration may affect the walls of a group of capillaries to such a degree as to interfere with the nutrition of portions of the brain. This condition, however, must not be confounded with the fatty degeneration which affects the capillaries as a result of softening of the brain.

These different conditions of obstruction of the circulation cause various lesions in the brain.

1. If the circulation is entirely cut off by the occlusion of a vessel of some size, or by occlusion or fatty degeneration of a group of capillaries, a portion of the brain tissue is found to be unchanged in color, but of soft or even fluid consistence. This alteration affects circumscribed portions, usually of the white substance of the hemispheres. These spots may be of all sizes, even the entire half of the cerebrum may be softened after ligature of one carotid. The softened tissue is composed of degenerated nerve fibres, large globules of myeline, compound granular corpuscles, corpora amylacea, concretions of lime, fatty ganglion cells, and lymphoid cells in variable number. The vessels are empty, normal, or fatty and broken into fragments.

The changes in these cases, often called "white softening," are, therefore, simply degenerative and necrobiotic, or of a very low inflammatory type. Such a spot of softening may remain unchanged for years, or it may become completely fluid and form a cyst with thick walls.

2. If a small artery is plugged, or there is thrombosis of a vein, the results are entirely different. In such cases, we find the appearances belonging to the hemorrhagic infarctions of all the viscera. The set of capillaries belonging to the artery or vein affected are distended with blood, a certain number of them rupture and allow the escape of blood into the tissue. The tissue around and between the distended capillaries and extravasated blood becomes the seat of degenerative and inflammatory changes. The portions of tissue affected are single or multiple, usually small, most frequently near the surface of the hemispheres. The brain tissue is red, mottled red and white, gray or yellow; it is soft or even puriform. It is composed of broken-down nerve fibres, globules of myeline, pigment masses, compound granular corpuscles, granular matter, lymphoid cells, fibrine or serum, and red blood globules. The blood-vessels are filled with blood, dilated, and often fatty.

After a time, these spots of so-called "red softening" may become fairly purulent, or may, from the loss of blood pigment, become yellow and gray-colored, or may become sclerosed, or rarely cheesy.

Abscess of the Brain.—When inflammation of the brain tissue goes on to an excessive formation of pus, abscesses are formed. A recent abscess has an irregular shape; its walls are composed of shreddy, disintegrated brain substance, and around it for some distance the brain tissue is softened. The pus is usually green and fetid. An old abscess becomes inclosed in a fibrous capsule, smooth inside, continuous with the brain outside; often very thick. The pus is always ill-formed, and may change into serum alone, so that the abscess looks like a cyst.

Fractures of the skull, either depressed or not; blows on the skull, without fracture; inflammation of the cranial bones or membranes; inflammation of the internal ear, with or without caries of the temporal bone, and pyæmia, are the usual causes

of abscesses. The abscesses may occur in any part of the brain, be of any size, and be solitary or multiple. They are always fatal, but may exist for years before death ensues.

Yellow Softening.—Under this name Rokitansky describes a peculiar condition of the brain. It occurs in circumscribed spots; has a moist, tremulous consistence, and a yellow color. The brain substance around is much swollen and oedematous. This form of softening may occur in any part of the brain. It is either idiopathic or secondary. It is found near hemorrhages and tumors.

Sclerosis.—Inflammation of the brain tissue, instead of rendering it soft, may cause induration.

As a senile change, the consistence of the brain often becomes harder and dryer. In Typhus, Cholera, puerperal Peritonitis, and Scarlet Fever, the brain may be firmer than usual, and contain less serum. In lead poisoning, the brain may be both indurated and atrophied.

In general paralysis, there is an induration of the superficial layers of the brain accompanying chronic meningitis.

In two cases of mild insanity of short duration, with paralysis of all the limbs, I have seen the entire white substance of the brain very markedly indurated. There was also a peculiar, colloid-looking degeneration of the gray matter of the cord.

Sclerosis of isolated portions of the brain (*Sclérose en Plâques*) occurs usually in connection with the same lesion of the cord. We may find it in any part of the white substance of the brain. The sclerosed patches are of variable size, number, and shape. They are firm, depressed, and shrunken, of a rosy or gray color. This lesion is characteristic of paralysis with tremor.

In all cases of sclerosis the essential process is an increase of the neuroglia, with atrophy of the nervous elements.

Interstitial Encephalitis.—Under this name Virchow has described a lesion which is very frequently found in still-born and very young children. It consists in an enlargement and fatty degeneration of the cells of the neuroglia of the white substance of the hemispheres, so that they look like compound granular corpuscles. It is still uncertain whether this change is to be looked upon as a pathological condition.

CEDEMA.

Infiltration of the brain with serum occurs as a consequence of chronic diseases—chronic nephritis, phthisis, etc.—and occurs with cardiac disease and obstruction of the jugular veins.

Usually the amount of serum is small; the brain substance appears moist, and exudes a little serum on pressure. Sometimes, however, the serum is very abundant, and the brain appears almost semi-fluid.

Edema, with anæmia of the brain, is considered by Traube to be the cause of uræmic convulsions.

MELANÆMIA.

In persons affected by malarial disease, the gray matter of the brain is sometimes found to have a blackish tint. This color is due to the presence of black pigment granules within the capillary vessels. The obstruction to the vessels by these pigment masses may cause capillary apoplexies.

The pigment is also found within the capillaries of the pia mater. Some authors hold that the pigment is situated in the walls of the vessels. It has always appeared to me to be within them.

NEW GROWTHS.

Carcinoma occurs in the brain as a primary or secondary tumor. It may be of the hard or soft varieties, or cavernous, or melanotic. It is found in all parts of the brain, and may be single or multiple. It may increase so as to involve the dura mater and skull, and appear on the outside of the cranium.

Sarcoma is usually found in the hemispheres. The tumors may be hard and fibrous, or soft, or vascular, or cheesy. They are composed of round, oval, or fusiform cells, with a connective tissue stroma.

Glioma.—Under this name Virchow describes a form of tumor composed of neuroglia. The typical growths are composed of a finely granular substance, in which nuclei are obscurely seen. They are either hard, or soft, or hæmorrhagic. They frequently appear like a hypertrophy of the brain tissue rather than a tumor.

Osteoma occurs in the form of isolated, globular tumors in the cerebrum and cerebellum, or has rather the character of an ossification of part of the brain. In these latter cases, it seems to be a late stage of the process of sclerosis.

Myxoma.—These tumors are found most frequently in the cerebral hemispheres. They are usually of large size, and of soft, gelatinous consistence, so as even to resemble cysts.

Fibroma is very rare. It occurs in the form of small, hard, globular tumors.

Cholesteatoma is found in the form of rounded tumors, having a fibrous capsule and a glistening, white consistence. They are made up of crystals of cholesterine and epithelial cells.

Angioma may occur in any part of the brain, but is most frequent in the floor of the fourth ventricle.

Tubercle is found most frequently in the cerebellum, and usually in children. The tumors may be single or multiple, large or small. They are usually globular, and have a concentric appearance; the greater portion often undergoes cheesy degeneration; but there is sometimes a layer of gray, semi-translucent material at the periphery.

Gummata.—Syphilitic tumors are found usually near the surface of the brain. They have an irregular shape, the larger tumors being made up of a number of small ones. They have a mottled gray and yellow color. Isolated portions undergo cheesy degeneration, and are separated by other portions, which are grayish and composed of round and fusiform cells, or white and firm, and composed of fibrous tissue.

New Growth of the Gray Matter of the brain, in the form of isolated nodules imbedded in the white substance, has been described by Virchow and others.

Cysts and dermoid cysts have been seen in the brain, but are rare. All the tumors of the brain have a tendency to cheesy and fatty degeneration, which renders their anatomical diagnosis difficult.

PARASITES.

Echinococci and *Cysticercus cellulosæ* are both found in the brain.

THE PITUITARY BODY.

The pituitary body is sometimes entirely absent, or it may be atrophied and fatty. Small abscesses have been observed in it. Colloid degeneration may affect it, sometimes with hypertrophy. Carcinoma and Tubercle may occur in it.

THE PINEAL GLAND.

This little body may be entirely wanting, or hypertrophied, or contain pus, or be changed into a cyst, or be enlarged and filled with globular concretions of lime. Förster reports a single case of secondary Carcinoma in it.

THE SPINAL CORD.

THE MEMBRANES.

THE DURA MATER.

Inflammation of the dura mater is very infrequent. It is only found with injuries, or disease of the vertebræ. The inflammation is usually of suppurative character, and may compress the spinal cord or cause a general meningitis.

NEW GROWTHS.

Bone of new formation in the dura mater is rare, and forms small plates.

Lipoma has been observed in several cases.

Tubercle usually accompanies caries of the vertebræ in the shape of a diffused, cheesy infiltration, with softening and the formation of abscesses between the pia and dura maters. More rarely, isolated tubercles are found without disease of the bone.

Carcinoma of the dura mater may be primary, or continuous from the vertebræ. It is medullary, grows inward and compresses the cord, or outward, and infiltrates the soft parts outside the vertebræ.

THE PIA MATER.

The pia mater of the cord, like that of the brain, is found in various degrees of congestion, and it is difficult to pronounce how great a degree is pathological.

Hæmorrhages occur idiopathically, or from injuries, or from general convulsions.

Serum is present beneath the pia mater to a greater or less degree. It is difficult to say how large an amount is pathological.

INFLAMMATION.

Spinal meningitis is found at all ages. It may be idiopathic, traumatic, rheumatic, or caused by inflammation of the brain, the dura mater, or the vertebræ, or occur as epidemic cerebrospinal meningitis.

The disease usually attacks the entire length of the membrane. An exudation composed of serum, lymphoid cells, and fibrine infiltrates the pia mater and raises it up from the cord.

NEW GROWTHS.

Fibroma of the pia mater, compressing the cord, has been observed.

Bone, in the form of small plates, sometimes occurs.

Carcinoma may occur as circumscribed tumors, or as diffused masses.

MALFORMATIONS.

Hydrorachis, Spina Bifida, consists in dropsy and atrophy of the cord, with or without malformation of the vertebræ.

(1.) The vertebral canal is open on its posterior surface for its entire length; the cord is absent, the nerves are inserted into the spongy hæmorrhagic membranes.

(2.) The vertebral canal is closed. The central canal of the cord is dilated with serum, or the cord is absent, and the membranes are dilated with serum; or there may be an accumulation of serum beneath the pia mater, with or without atrophy of the cord.

(3.) At some portion of the vertebral canal its posterior wall is absent, so that the membranes protrude as a sac, filled with serum—Spina Bifida. The serum accumulates in the central canal, or beneath the pia mater. The cord is absent, or rudimentary, or well formed. The walls of the sac are formed of dura and pia mater, or of pia mater alone. Rarely there is no fissure in the bones, and the sac protrudes between the laminar

processes. In rare cases, there is a defect in the anterior wall of the canal, and the sac projects through it. I have made an autopsy on one very remarkable case of this kind. The patient was under the care of Dr. Emmet.

A female, aged 36, ten years ago noticed a swelling as large as a goose's egg in the left iliac region, attended with severe pain and constipation. This swelling increased slowly. Seven years ago, she had a bloody and purulent discharge from the bowels, lasting for five weeks, and followed by a decrease of the swelling. Was seen by Dr. Emmet about three weeks before her death. There was then œdema of the right leg, pain in the sacrum, constipation of the bowels, urine normal, abdomen tympanitic. The cervix uteri was high up; the rectum was nearly occluded by a fluctuating tumor posterior to it. From this tumor a little clear serum was drawn off with a fine trochar. On the day after this operation, she had a chill, followed by fever, great prostration, and in seven days, death.

Autopsy 9½ hours after death. The rectum and descending colon were enormously dilated with feces, nearly filling the abdomen. The upper edge of the uterus was two inches below the umbilicus, and pushed to the right side. The bladder was also carried up in front of the uterus. There was cystitis. There was a large cyst behind and to the right of the rectum, filling the pelvic cavity, and reaching up to the level of the second lumbar vertebra. This cyst was firmly bound down on all sides, but especially at the lower part of the sacrum. When it was removed, there was found an opening in the lower part of the anterior surface of the sacrum, through which the finger could be passed into the spinal canal.

(4.) A portion of the cord may project through an opening in the bones, without any serum. Isolated cases have been reported of abnormally short cord, and of a double cord.

INJURIES.

The spinal cord may be compressed or lacerated by penetrating wounds, by fracture, or dislocation of the vertebræ, or by concussion, without injury to the vertebræ. The spinal cord is found simply disintegrated, or hæmorrhagic, or if inflammation

has followed the laceration, lymphoid cells and compound granular corpuscles will be present. There may be marked changes in the minute structure of the cord, without any evidence to the naked eye.

HÆMORRHAGE.

Apoplexies in the substance of the cord are not common. The nervous tissue is disintegrated by the blood. The lesion may cause death in a very short time, or may be followed by myelitis.

INFLAMMATION.

Myelitis may be traumatic, idiopathic, or secondary. It occurs under so many different conditions, that it is difficult to speak definitely of its causation. It produces either softening or hardening of the cord.

(1.) The cord is broken down, of a cream-like consistence, of a reddish or yellow color. The softening may affect the entire thickness of the cord, or be confined to certain parts; it may extend the entire length of the cord, or be confined to certain regions. It seems to commence usually in the gray matter. The softened portions are composed of broken-down nerve tissue, blood globules, lymphoid cells, and compound granular corpuscles. The cord may also be softened without any change in its color, and then consists merely of fine granular matter, with a few compound granular corpuscles.

(2.) Myelitis causing induration, (sclerosis,) may affect the entire organ or circumscribed portions. The diffuse form renders the cord anæmic and hard throughout.

The circumscribed form is identical with the *scélérose en plaques* already described in the brain.

Locomotor Ataxy.—In this disease, part of the white substance of the cord undergoes a peculiar degeneration. The posterior columns are usually affected. We find them changed, in the earlier stages, into a reddish gray, soft substance. This change is produced by an increase of the neuroglia and an atrophy of the nerve fibres. At a later stage, the affected portions are firmer and shrunken. It is a form of sclerosis.

NEW GROWTHS.

Carcinoma, Sarcoma, Tubercle, and Syphilitic Growths have all been observed in the cord. They resemble closely the same varieties of tumor in the brain.

A very remarkable form of new growth on the cord has been shown to me by Dr. E. C. Seguin. The patient was a woman, thirty years of age. Two years before her death, she received a severe blow on the back of the head. After this, first one side and then all the limbs became paralyzed. From this condition she recovered sufficiently to walk; but after a short time, she again grew worse, and both arms and legs were paralyzed and contracted. She died of bronchitis. At the autopsy there were found two tumors on the spinal cord within the dura mater.

The upper tumor was on the anterior face of the cord just below the decussation of the medulla. It was of ovoid shape, and of the size of a pigeon's egg. It was composed of branched connective tissue with many round cells.

The lower tumor was on the posterior face of the cord, at the upper part of the lumbar enlargement. It was of the size and shape of a large, flattened cherry. It was composed of portions of two spinal cords fused together, with their long axes parallel to that of the normal cord. There were four patches of gray matter, having the shape of the cornua, containing ganglion cells, and joined two and two by regular gray commissures, in each of which was a central canal. These gray portions were surrounded by tissue resembling the normal white substance of the cord.

THE RESPIRATORY SYSTEM.

THE LARYNX.

MALFORMATIONS.

THE larynx may be entirely absent in acephalous, and other monsters. It may be abnormally large, or abnormally small. The epiglottis may be larger or smaller than the normal standard, or may be cleft. Portions of the cartilages of the larynx may be absent or defective.

INFLAMMATION.

Acute Catarrhal Laryngitis causes the mucous membrane to appear congested, swollen, and soft. Its surface is thinly coated with muco-pus. The mucous glands are swollen with their increased secretion. The submucous connective tissue is more or less infiltrated with serum.

Chronic Catarrhal Laryngitis causes the mucous membrane to appear grayish and soft. The mucous follicles are swollen and prominent. The submucous tissue is thickened. The surface may be covered with muco-pus, and there are often small ulcerations. Sometimes the inflammation extends to the perichondrium of the cartilages.

Croupous Laryngitis occurs as the "croup" of children, with diphtheria, pyæmia, typhus fever, and other severe, acute diseases, and from the inhalation of irritating substances and gases. We find the mucous membrane covered by a layer of yellowish-white, consistent material—false membrane. This layer consists of fibrine and lymphoid cells; it dips down into the mouths of the mucous follicles, and may be found also in

the submucous tissue. The exudation adheres firmly to the epithelium of the part, and when, after a time, it becomes loosened and falls off, the epithelium goes with it.

The diagnosis between diphtheria and the croup of children belongs to clinical medicine. The anatomical lesion of diphtheria is a croupous exudation on and in whatever mucous membrane the disease happens to show itself.

Inflammation of the Submucous Connective Tissue occurs as :

(1.) Œdema glottidis. Here we find the epiglottis swollen, and from its base two bags of serum, formed of the aryteno-epiglottidean folds, extend backward. This œdema is often sufficient to close the larynx and cause death. It accompanies acute catarrhal laryngitis, small-pox, typhoid fever, syphilitic and tuberculous ulcerations, facial erysipelas, and perichondritis.

(2.) Purulent inflammation may attack the same parts which are implicated in œdema glottidis, and cause death in the same way.

Perichondritis.—Inflammation of the perichondrium of the cartilages of the larynx may be idiopathic or secondary to inflammations of the mucous membrane. It usually attacks first the cricoid cartilage, and extends later to the other cartilages. The inflammation causes the production of pus and separation of the perichondrium from the cartilage, with the death of the latter. The cartilage is usually found ossified, sometimes carious, sometimes degenerated and softened without ossification. The pus spreads to the submucous tissue, collects in abscesses, and may cause death by suffocation, or may perforate the mucous membrane. When the inflammation extends outward, abscesses and sinuses form on the outside of the larynx, and may perforate through the skin of the neck. Portions of the necrosed cartilages may find their way into the cavity of the larynx and be coughed out, or may become fixed in the rima glottidis and cause suffocation, or may pass into the bronchi. In other cases, the portions of cartilage may be discharged outward through openings in the skin.

Syphilitic Laryngitis.—Constitutional syphilis shows itself very frequently in the larynx. It produces here simple catarrhal inflammation, or the mucous follicles may swell and ulcerate, or

the little lymphatic follicles in the submucous tissue may swell and ulcerate. The syphilitic condylomata produce much graver lesions. They form small, rounded nodules on the surface of the mucous membrane. These nodules may become aggregated together so as to form masses of considerable size, and of soft, medullary consistence. Either the large or the small nodules may ulcerate. The ulceration, if extensive, may induce perichondritis, and also hypertrophy of the mucous membrane near the ulcerations. We find then the cavity of the larynx often entirely deformed by a series of changes which have caused destruction of tissue in some places and hypertrophy in others.

Tubercular Laryngitis usually accompanies pulmonary phthisis. In patients affected by phthisis we often find in the posterior wall of the larynx, between the arytenoid cartilages, one or more small, deep ulcers with overhanging walls, whilst there is often at the same time catarrhal laryngitis. In more severe cases, these little ulcers are numerous and become joined, or they are from the first of large size. In such cases, we may also have papillary hypertrophies of the mucous membrane and perichondritis.

It is still uncertain how far these lesions are to be ascribed to submucous tubercles, and how far to follicular ulceration.

NEW GROWTHS.

Ossification of the thyroid and cricoid cartilages is a regular senile change. The same thing is often caused by perichondritis. The epiglottis may become calcified, but does not change into true bone.

Mucous Polypi are small, soft, rounded tumors with a pedicle. They are composed of a fibrous stroma covered with a thick layer of epithelium. Properly speaking, they are examples of papillary Fibroma.

Fibrous Polypi usually occur in the upper part of the larynx, and may reach the size of a pigeon's egg. They are attached to the larynx by a broad or narrow pedicle, and are composed of fibrous tissue covered with epithelium.

Chondroma.—Cartilaginous tumors grow from the thyroid

and cricoid cartilages as flat or globular tumors. They usually grow inward, and may be mistaken for polypi. They may become ossified.

Epithelial Cancer may appear in any part of the larynx. It forms irregular, fungous, ulcerating masses, projecting into the cavity of the larynx. The new growth may also involve the parts about the larynx and form tumors in the neck. These external tumors are often surrounded by inflamed and suppurating tissue.

Carcinoma as a primary disease seldom occurs in the larynx. It forms nodules in the submucous tissue, which may enlarge, project inward, and ulcerate.

Sarcoma occurs in the form of polypoid tumors growing from the submucous tissue.

PARASITES.

In the catarrhal and croupous exudations of the larynx, the spores and thallus of fungi are often found. Some authors regard them as the cause of diphtheritic inflammation.

THE TRACHEA.

MALFORMATIONS.

The trachea may be entirely absent. It may communicate with the œsophagus. There may be little fistulous canals opening from it, which penetrate the skin of the neck. The cartilages may be increased or diminished in number, or parts of them may be wanting. The trachea may divide into three bronchi instead of two. It may be misplaced on the left side of, or behind the œsophagus.

ALTERATIONS IN SIZE.

A uniform dilatation of the trachea is sometimes seen in old persons as a consequence of long-continued catarrhal inflammation.

Rokitansky describes a peculiar dilatation of the posterior wall. The ends of the cartilages are widely separated, the mu-

cous membrane appears pushed outward into numerous small sacs, separated by bands of muscle.

Stenosis of the trachea may be produced by thickening from chronic inflammation, by ulceration and cicatrization, and by the pressure of tumors from without.

INFLAMMATION.

Inflammation of the trachea has the same characteristics as, and almost always accompanies that of the larynx and bronchi.

NEW GROWTHS.

Ossification of the tracheal cartilages is a senile change.

Carcinoma is secondary, usually, to cancer of the œsophagus. A single case of primary carcinoma of the trachea and bronchi is described by Langhaus. There were small flattened nodules in the submucous tissue.

Lymphoma in the submucous tissue at the lower end of the trachea has been seen in leucæmia.

Chondroma appears usually in the form of small multiple tumors growing from the inner surface of the tracheal cartilages. A number of little tumors may grow so close together that they become joined and form a sort of network. They may be so loosely attached to the cartilage that they appear to be free in the submucous tissue. They may become ossified.

THE BRONCHI.

INFLAMMATION.

Acute Catarrhal Bronchitis occurs at all ages; in childhood and old age, it is apt to attack the smaller bronchi; in adult life, usually the larger. The mucous membrane appears swollen, soft, red, and covered with mucus or muco-pus, the pulmonary tissue is at the same time usually congested and somewhat œdematous. In children, capillary bronchitis is very frequently accompanied by peri-bronchitis, lobular pneumonia, and atelectasis. The lobules of pneumonia are of very variable size, and the lung tissue between them may be anæmic. The atelectasis

is produced by stoppage of the bronchi and consequent non-aeration of portions of lung tissue. The portions so affected are dense, firm, non-aerated, of a deep purple color, the section is smooth and dry, resembling foetal lung. Portions of lung in a condition of atelectasis may become inflamed, and thus a complicated condition be produced.

Chronic Catarrhal Bronchitis is usually a disease of adult life. The mucous membrane of the bronchi is of a deep red color, or pale, or grayish, soft, thickened or thinned, granular or reticulated. This reticulated or trabeculated appearance is found in the lungs of persons subject to long-continued cough. The longitudinal and transverse fibrous bands of the bronchial wall become separated, the tissues between them atrophied, so that the wall assumes a trabeculated appearance. When the bronchi are cut across, muco-pus exudes from them. It is very common to find the lungs emphysematous, or with some degree of fibrous induration, or congested and oedematous. In some cases the exudation becomes foul and stinking, and the walls of the bronchi black and gangrenous. Sometimes the exudation is entirely purulent, or of a greenish watery character. It is expectorated in large quantities, and causes gradual emaciation and death of the patient. Especially in these last cases we find dilatation of the bronchi. Chronic bronchitis and emphysema may give a clinical history very closely resembling that of phthisis. They are sufficient of themselves to cause death, and even sudden death.

From the obstructions in the bronchi, the air-cells may be abnormally dilated and the whole lung expanded: this condition is not to be confounded with emphysema.

Croupous Bronchitis occurs in children in connection with croupous laryngitis and tracheitis. The larger bronchi, and more rarely the smaller, are lined or filled with a mass composed of coagulated fibrine and lymphoid cells. Sometimes the exudation will be continuous through a set of bronchi and a complete cast of them will be coughed up.

In adults, the disease is idiopathic, or a complication of croupous pneumonia. When idiopathic, it may run an acute course, or last for months and years, with repeated expectorations of

casts of the bronchi. The chronic form affects only a few of the bronchi. The mucous membrane will be found congested or anæmic. The lungs are usually diseased; either emphysema, or fibrous induration, or small apoplexies, or abscesses are present. The idiopathic form in adult life is rare.

Croupous pneumonia is always attended with croupous exudation into the small bronchi, and in some cases the larger bronchi also will be found partly filled with long, whitish cords of the same material.

Peri-Bronchitis.—Inflammation with the formation of lymphoid cells and connective tissue in the walls of the bronchi and just around them is found as an acute and chronic condition. It not infrequently occurs in the acute pneumonia of children, and we see the hepatized lung mottled with white lines and spots. In the chronic inflammation of phthisis, we usually find the walls of the bronchi thickened, sometimes enormously so.

BRONCHIECTASIA.

Dilatation of the bronchi presents itself under three forms, the cylindrical, the fusiform, and the sacculated.

The cylindrical form consists in the uniform dilatation of one or more bronchi. It is found in bronchi of every size, but most frequently in those of medium diameter. The large and medium-sized bronchi may be dilated and the smaller remain normal, or the terminal bronchi alone be affected.

The fusiform dilatation is a mere variety of the cylindrical. The bronchus is uniformly dilated for a short distance, and then resumes its normal size. Several such dilatations may be found in the same bronchus.

The sacculated dilatations form the largest cavities. These cavities usually communicate with the bronchus only on one side, the peripheral portion of the bronchus becoming obliterated. The bronchus leading to the cavity may be of normal size, or dilated, or stenosed, or even completely obliterated, so that there is no opening into the bronchiectasia.

These sacculated dilatations may attain to a very large size, and may be so numerous that the entire lung looks honey-combed.

The walls of these bronchiectasiæ are found in various morbid conditions, but usually preserve something of their normal structure.

(1.) The wall is simply swollen and relaxed. This condition is most frequent in the dilatations caused by bronchitis in children. As the bronchitis subsides, these dilatations may disappear.

(2.) The wall is hypertrophied. The mucous membrane is thickened, and may be covered with little papillary outgrowths. The submucous tissue, mucous glands, and cartilage may also be hypertrophied.

(3.) The wall is atrophied, and the thinning may be so advanced as to leave nothing but an attenuated, semi-transparent mucous membrane.

(4.) The wall is trabeculated. The surface is no longer smooth, but is divided up by bands crossing each other, and leaving little depressions between them. These bands are the longitudinal and circular fibrous bands of the normal bronchi. The cartilages are usually thickened.

(5.) They may ulcerate, and this ulceration may extend to the lung tissue, so as to form cavities, which are partly bronchiectasiæ, and partly formed by the destruction of lung tissue.

(6.) The bronchus leading to a dilatation may become entirely obliterated, and leave a cyst with smooth walls and serous contents.

(7.) The walls may become calcified, or inflamed, or gangrenous.

The lung tissue will be found altered in various ways, and these alterations either precede, or follow, or are complications of the dilatations.

(1.) The lung tissue is in a condition of fibrous induration. This fibrous induration may precede and cause the dilatations, or may be with them the result of chronic inflammation.

(2.) The ectasiæ may give rise to lobular pneumonia and congestion. The lobules affected by pneumonia may be in the condition of red and gray hepatization, or may become gangrenous.

(3.) A moderate degree of emphysema is a frequent concomi-

tant of bronchiectasia; higher degrees are not so common, and are more apt to occur in other parts of the lung.

(4.) Old pleuritic adhesions are very frequently found, and are regarded as one of the most common causes of bronchiectasiæ.

(5.) In a few rare cases, pneumothorax has been caused by perforation of a superficial bronchial dilatation.

The situation of bronchiectasiæ depends upon the previous disease of the lungs or pleura. Capillary bronchitis, catarrhal pneumonia, and atelectasis cause dilatations of the bronchi, in the inferior and posterior portions of the lung. Chronic inflammation, phthisis, and fibrous indurations cause dilatations principally in the upper lobes. Old pleuritic thickenings and adhesions cause dilatations in the portion of lung which they cover. Taking all the cases together, the lower lobes are the most frequently affected. Both lungs are as frequently involved as one. The largest bronchi are the ones least frequently involved.

STENOSIS.

Narrowing of the bronchi is produced either by the compression of external bodies, or by the contraction of their walls.

External pressure is produced by aneurisms of the aorta, enlarged lymphatic glands, abscesses and new growths of the mediastinum, tumors of the œsophagus, and cancer of the lungs.

Internal contraction is produced by (1) ulceration and cicatrization, usually of syphilitic origin; (2) inflammatory thickening of the bronchial walls; this is a very rare occurrence; (3) poly-poid new growths; these also are very rare.

Stenosis of the bronchi very often produces dilatation of the bronchi beyond the stenosis, lobules of emphysema, or of atelectasis, capillary bronchitis and pneumonia.

NEW GROWTHS.

Ossification of the bronchial walls has been seen in a few rare instances.

Carcinoma is secondary to the same new growth in the lungs, mediastinum, or œsophagus. A single case of primary carcinoma of the trachea and bronchi is given by Langhaus.

Lipoma in the submucous tissue has been seen in one case by Rokitansky.

THE LUNGS.

MALFORMATIONS.

One or both lungs may be entirely wanting, or only partially developed.

A peculiar degeneration, by which the lung is converted into a number of sacs containing air and serum, and communicating with the bronchi, has been seen in a few instances.

The lobes may be subdivided by deep fissures. An accessory lobe, separated from the lung, has been seen between the base of the left lung and the diaphragm.

There may be hernia of the lung, with absence of part of the thoracic wall.

There may be transposition of the lungs, with the same change in the position of the heart and liver.

In rare cases, the congenital smallness of the lungs remains, and persons continue to live for a number of years.

INJURIES. PERFORATIONS.

Contusions of the thorax may cause rupture of the lung tissue and fatal hæmorrhage. The lungs may be wounded by projectiles and penetrating instruments of all kinds, and by fragments of bone. Even severe injuries of this nature may not prove fatal.

Collections of pus in the pleural cavities and mediastinum, and abscesses of the liver, spleen, and kidneys, may perforate the lungs.

Fistulous openings from the lungs through the thoracic wall may be formed during phthisis, emphysema, gangrene of the lungs, abscesses, hydatids, and wounds.

CONGESTION AND OEDEMA.

These conditions are found in the dead body not only as post-mortem changes, but also as evidences of the same conditions during life.

Congestion and œdema confined to the posterior portions of the lower lobes are usually merely post-mortem changes, and are most marked in hot weather. When, however, the congestion and œdema are excessive, and especially when they involve the upper lobes, we may conclude that they existed during life. To determine, however, their importance in producing death, a reference to the clinical history is necessary. Congestion and œdema, in different relative proportions, occur with cardiac disease, with changes in the nervous centres retarding the act of respiration, with coma from any cause, with renal disease, with any chronic, exhausting disease, general anasarca from any cause, bronchitis and pneumonia, narcotic poisons and sunstroke.

In some of these cases, as in renal disease, general anasarca, bronchitis and pneumonia, the congestion and œdema appear to be the direct cause of death. In exhausting diseases, also, the case is the same, although here there is very often filling up of the air-cells with the products of inflammation.

In persons dying comatose, however, from any cause, the condition of affairs is different. The congestion and œdema, although often very excessive, are merely the accompaniments of diseases which are of themselves sufficient to destroy life.

HÆMORRHAGE.

Effusions of blood which lacerate the lung tissue are not common. They are produced by penetrating wounds, contusions, atheroma, and dilatation of the pulmonary arteries, rupture of aortic aneurisms, and rarely by a simple effusion of blood into the alveoli. We find a considerable portion of the lung tissue broken down and infiltrated with blood, and the blood may also rupture through the pleura.

Cavities in the lungs produced by phthisis, syphilis, or gangrene are sometimes found filled with blood from the ulceration of a large vessel.

The most common form of hæmorrhage, however, is that called "pulmonary apoplexy," or hæmorrhagic infarction. This consists in the filling up of the air-cells with blood. We find at any portion of the lungs, and even in lungs compressed by pleuritic fluid, a few nodules, from the size of a chestnut to that

of a hen's egg. These nodules are firm and of a dark red color. The lung tissue around them is hyperæmic, or fairly in the condition of red or gray hepatization. Minute examination shows the alveoli, bronchi, and blood-vessels in these nodules to be completely filled with blood globules. These infarctions may cause death by their size and number, or by the pneumonia and pleurisy which they excite. In other cases, the blood globules break down and become fluid, are partly absorbed, partly coughed out, and partly changed into pigment. The air-cells which had been filled up may again be expanded with air, or may contract and form a spot of fibrous induration. In still other cases, the lung tissue becomes gangrenous and forms cavities with irregular walls and brownish semi-fluid contents.

Hæmorrhagic Infarctions also form the most constant lesion of pyæmia, although with a somewhat different history. They are usually situated near the surface of the lung, are small and often numerous. At first they resemble the non-pyæmic infarctions, and look like dark red nodules. Very soon, however, there is a narrow zone of pneumonia around them, then their centres become decolorized, soft, and white; soon this softening involves the entire infarction and forms a small cavity filled with whitish puriform fluid.

The causation of these pyæmic infarctions is very clear. They are produced by the plugging of a small artery with an embolus, and this embolus is usually part of a broken-down thrombus in some vein situated in inflamed tissue. It is the necrotic and unhealthy character of the embolus which gives to the infarction its tendency to rapid decomposition and suppuration.

The non-pyæmic infarctions are in some cases undoubtedly produced by the same mechanism. The embolus then comes from vegetations in the right cavities of the heart. Whether such infarctions are always produced by emboli is still uncertain.

EMPHYSEMA.

Emphysema is of two kinds, vesicular and interlobular.

(1.) *Vesicular Emphysema* consists in an abnormal dilatation of the alveoli of the lung, with thinning and atrophy of their walls.

We must distinguish from this a simple dilatation of the alveoli, which we find dependent on various conditions. The bronchi may be partially obstructed so that the escape of air is impeded, and after death the lungs remain unnaturally inflated. Or a considerable portion of the lung tissue is rendered impervious to air by pneumonia, oedema, or pleurisy, and the rest of the lung is unnaturally dilated. Or, the walls of the alveoli may lose their elasticity so that they no longer expel the air, but remain dilated.

In true vesicular emphysema, we find the lungs pale, anæmic, large, of a soft cushiony texture, the alveoli distinctly visible with the naked eye. The walls of the alveoli are thinned, the capillary vessels are small, and the spaces between them are increased in size. Often a portion of the walls of the alveoli entirely disappear, so that several alveoli become fused into one. These changes are apt to be most marked at the edges of the lobes. This change in the lung tissue may affect both lungs, or one lung, or part of a lung. Very often when part of a lung is contracted and indurated, the neighboring portions will be emphysematous.

Emphysema is almost always associated with some degree of chronic bronchitis. It also leads to hypertrophy of the right ventricle of the heart, to expansion of the walls of the thorax, and to displacement downward of the diaphragm and the abdominal viscera.

Emphysema and chronic bronchitis may of themselves cause death. In other cases, the lungs become the seat of an acute croupous pneumonia, and this causes death, often when only part of a single lobe is hepatized. Fibrous induration, either in bands or nodules, is also sometimes associated with emphysema.

Interlobular Emphysema consists in the escape of air from the alveoli into the interstitial connective tissue, or beneath the pleura. In the latter situation, it may form vesicles of considerable size. This form of emphysema is not of very frequent occurrence, nor of much pathological importance. It may be produced by any thing which causes rupture of the alveoli. In rare cases, there may be rupture of the pleura and pneumotho-

rax, or the air may pass into the mediastinum, and from thence upward into the neck, producing general emphysema.

ATALECTASIS.

A collapsed and unaerated condition of portions of lung tissue is either congenital or acquired.

(1.) In congenital atelectasis we find portions of the lungs firm, non-crepitant, of a dark blue or purple color, depressed, smooth on section, and containing a variable amount of blood. These portions can usually be artificially inflated, and then cannot be distinguished from the surrounding pulmonary tissue. This condition is produced by the inability of the child after birth to fully inflate its lungs; either from want of sufficient vitality, or from mechanical obstruction in the bronchi. If the child lives for some time, and the collapsed lobules do not become inflated, they become hard, and can no longer be inflated.

(2.) Young children may acquire the same condition as the result of bronchitis. A small bronchus becomes obstructed, and the air-vesicles which it supplies collapse. We then find, scattered through the lungs, collapsed lobules resembling those found in the new-born child. There may be at the same time lobules in a condition of inflammatory hepatization; and the collapsed lobules may also become inflamed.

(3.) In adults, large or small portions of lung tissue may become collapsed, as the result of bronchitis; of stenosis of a large bronchus; of paralysis of the pneumogastric; of atrophy of the respiratory muscles; of compression of the lungs by fluid, by new growths, or by thickened pleura. The collapsed portions are unaerated, firm, smooth; of a leathery consistence; of a dark red or blue color, and in various degrees of congestion. The names of "carnification" and "splenization" have been somewhat loosely applied to this condition.

PIGMENT INDURATION.

This change in the pulmonary tissue is one of the regular sequelæ of grave cardiac disease. Any disease of the heart which produces an obstruction to the free passage of blood from

the left auricle into the left ventricle may produce this lesion. Stenosis and insufficiency of the mitral valve, and stenosis of the aortic valve, are the cardiac lesions most frequently found.

The lungs appear small, the pleura somewhat thickened; the surface of the lungs is of a pale pinkish color, mottled with brown or red. Their cut surface is of a bright red color. They contain less air than usual, and have a firm, leathery texture. Very little blood exudes from their cut surface; they are sometimes dry, sometimes œdematous. Not infrequently there are lobules of hepatization and pulmonary apoplexies, or the greater part of a lobe may be hepatized.

The lesion consists in, (1,) dilatation of the capillaries; (2,) thickening of the walls of the air-cells; (3,) increase of pigment; (4,) an accumulation of large cells in the alveoli, as in a low grade of catarrhal pneumonia. This last is the most constant lesion, and the one of greatest pathological importance; for by it the alveoli become gradually filled up, and respiration is seriously impeded. The large epithelial cells produced by the inflammation often contain granules of dark pigment.

INFLAMMATION.

Inflammation of the lungs affects either the alveoli or the interstitial connective tissue. When it affects the alveoli, they become filled with inflammatory exudation; when the interstitial tissue is involved, it becomes increased in amount.

1. *Croupous Pneumonia* is one of the most common diseases of adult life. It usually involves the greater part of a lobe, and may extend over one or both lungs. It passes through three stages.

(1.) *Congestion*.—This stage usually escapes our observation in the human subject, but may be artificially produced in animals. The lung is dark colored, and exudes a large amount of blood and serum. The blood-vessels, both large and capillary, are gorged with blood; the alveoli and small bronchi contain a small amount of a material which is coagulated by alcohol into an amorphous, granular, or dimly fibrillated substance.

(2.) The first stage is soon followed by the second, that of Red Hepatization. The lung loses its spongy consistence, and

becomes solid. Firm pressure causes it to break down under the finger. The color is red, sometimes mottled with hæmorrhagic spots of a deeper hue. A section looks as if the lung tissue were composed of a multitude of small granules closely packed together; and these granules can be picked out with the point of a needle. If the lung is seen soon after death, the section is dry; in a short time, however, part of the exudation liquefies, and the section will be covered with a thick, grumous, reddish-gray fluid. The larger bronchi are not infrequently filled with firm, yellow, fibrinous cords, continuous with smaller cords in the smaller bronchi. Minute examination shows the alveoli and small bronchi to be filled up with a mass of new material, composed of fibrine coagulated into a delicate net-work, lymphoid (pus) cells, large polygonal nucleated cells, and red blood globules. The fibrine varies somewhat in amount, but is always present. The lymphoid cells are very numerous; some of them are as pale as perfectly fresh pus-cells; but most of them are granular and somewhat swollen. The large, polygonal, nucleated cells are identical with the cells found in very small numbers in the adult lung, and more abundantly in the infant lung. They appear to be epithelial cells. They are found in variable numbers, most abundantly in the pneumonia which attacks emphysematous and phthisical lungs. The red blood globules give the coloring to the mass. The walls of the alveoli are unchanged; the capillary plexus in the walls contains but little blood.

(3.) Finally comes the third stage, that of Gray Hepatization. The lung retains the same consistence, but is usually much moister; the section is covered with grumous fluid. The color changes from red to gray; not at once, but gradually, so that the lung is first mottled red and gray, and then is entirely gray. Minute examination shows the air-cells and bronchi still filled with new material; but this material has undergone changes. The red blood globules have vanished. The lymphoid cells and large cells remain, but are distended with shining granules. There is a considerable amount of granular matter formed by the breaking down of these cells and of the fibrine. This process of degeneration and disintegration of the elements of the

exudation goes on until the alveoli are filled with a fluid mass, which can be coughed out, and the pneumonia is resolved.

Sometimes, however, the change of color is not accompanied by changes in the exudation. A lung may be entirely gray—almost white—and yet the section is dry, the fibrine remains, and the cells are but little degenerated. This condition is sometimes found in persons whose lungs remain hepatized for an unusually long time without either resolution or death taking place. In most cases, the pulmonary pleura covering the hepatized lungs will be found covered with a thin layer of fibrinous exudation.

Croupous pneumonia may prove fatal at any stage; its fatality depending on the amount of lung hepatized, the general condition of the patient, and the amount of congestion and cedema of the rest of the lung tissue.

Sometimes abscesses form in the hepatized tissue. This takes place by a molecular degeneration and destruction of the walls of some of the air-cells, with an increased production of lymphoid cells and serum. Such abscesses may prove fatal of themselves, or may perforate the pleura, or may be absorbed, or may become concrete and calcified.

Gangrene of the lung, also, may result from hepatization, although in these cases there seems always to be thrombosis of the bronchial arteries.

The exudation in the air-cells, after reaching the stage of gray hepatization, may undergo a dry, granular degeneration. The air-cells are then filled with a firm, dry, yellow mass composed of granules and atrophied cells; and there is cheesy hepatization.

2. *Catarrhal pneumonia* differs from the croupous form in the character of its exudation. The material which fills the air-cells contains little or no fibrine, a smaller number of lymphoid cells, and a much larger number of the large, nucleated cells. The disease runs through the same stages of congestion: red, gray, and cheesy hepatization. The section is smoother, and without the granular appearance of croupous hepatization. There is a much stronger tendency to pass into the cheesy condition. Catarrhal pneumonia usually affects only lobules of the

lungs; sometimes only a few alveoli. There is always capillary bronchitis; and the small bronchi are filled with pus. There may also be peribronchitis.

In children under five years old, pneumonia as a rule is of the catarrhal variety. The pneumonia around pyæmic infarctions, in pigment induration, and in phthisis, is usually also catarrhal.

The attempt to class this form of inflammation of the lung with the catarrhal inflammations of mucous membranes is not altogether satisfactory. The alveoli of the lungs are not lined with a mucous membrane, nor does their inflammation ever have a true catarrhal character. All sorts of gradations, also, can be found between the croupous and so-called catarrhal exudations. The name is used, therefore, not as a good one, but for want of a better.

3. *Hypostatic Pneumonia*.—In persons who have been for a long time confined to bed by severe and protracted illness, we often find the lungs, especially the lower lobes, congested and œdematous. A careful examination will often discover, also, small, irregular patches of red hepatization. In these patches, the alveoli are filled almost entirely with lymphoid cells, hardly any fibrine being present.

4. *Interstitial Pneumonia*.—An increase of connective tissue fibres and cells is often seen in circumscribed portions of the lung, especially at the apex, either as the result of a lobular pneumonia or as a simple induration. With chronic bronchitis, emphysema, anthracosis, around abscesses, in syphilis, and with extensive pleuritic adhesions, it also occurs. Its most marked forms, however, occur in phthisis, and will be described with that disease.

In rare cases, there is suppurative inflammation of the interstitial tissue, forming thin layers of pus between the lobules of the lung, with abscesses beneath the pleura.

GANGRENE OF THE LUNGS.

Portions of the lungs may not only die, but may also undergo decomposition and become putrid. Such a gangrenous process may be caused by infarctions, by pneumonia, with throm-

basis of the bronchial arteries; and very often occurs without any satisfactory reason. The gangrene may be circumscribed or diffuse.

In the circumscribed form we find small nodules, usually near the periphery, changed into a brownish, putrid mass, sharply defined from the surrounding tissue. After a time, the periphery of the nodule becomes fluid, and there is a cavity filled with fluid and gangrenous tissue. The gangrene may extend to the pleura and cause acute pleurisy or hydropneumothorax. The gangrenous cavity may communicate with a bronchus and cause bronchitis; or a large vessel may be eroded and the cavity be filled with blood. Sometimes a circumscribed gangrene becomes the centre of a diffused gangrene. In rare cases, the cavity may contract and cicatrize.

Diffused gangrene may involve an entire lobe. The lung-tissue will be found changed into a moist, stinking mass. The neighboring lung-tissue is œdematous or hepatized, the pleura is inflamed or gangrenous.

PULMONARY PHTHISIS.

Under this name all physicians are well acquainted with a disease characterized by cough, mucous, purulent, or bloody expectoration, dyspnœa, hectic fever, gradual emaciation, and physical signs indicating condensation of the lung tissue, and exudation in the bronchi.

The lungs of persons who have suffered from this disease present, after death, a variety of lesions. These lesions it is convenient to group under three heads, according to their most marked anatomical condition, namely, Catarrhal, Fibrous, and Tubercular Phthisis. These three forms occur either separately or together.

1. *Catarrhal Phthisis*.—This form may run a rapid course or one of long duration. It may entirely resolve and leave no trace behind, or may invade and destroy the larger part of the lung tissue. The pathological process consists in a catarrhal inflammation affecting the alveoli and small bronchi. The pneumonia passes through the stages of congestion, red hepatization, and gray hepatization. After reaching the stage of gray

hepatization, the exudation undergoes a process of dry degeneration, converting it into a firm, dry, cheesy mass. This mass is usually of an opaque, yellow color, less frequently of a peculiar gray. The lung tissue is then said to be in a condition of cheesy hepatization. The portions of lung which pass into this condition of cheesy hepatization vary much in size. A large part of a lobe may be hepatized throughout; or there are large yellow lobules, the size of a hen's egg, scattered through the lungs; or there are great numbers of small yellow nodules, from the size of a pin's head to that of a pea, thickly studding the lung, often aggregated together, the lung tissue between them either anæmic, or normal, or congested, or hepatized. The larger masses are the infiltrated tubercles; the smaller, the miliary tubercles of Laennec. In all of them the lesion is the same: the alveoli and small bronchi are filled with exudation which has undergone cheesy degeneration. In the larger lobules, a great number of alveoli are thus filled up; in the miliary nodules, only a few alveoli or a small bronchus are hepatized.

In the lungs of children, we find a form of miliary hepatization in which a small bronchus or a few alveoli are filled with cheesy matter, and there is at the same time a production of round and fusiform cells in the wall of the bronchus or of the alveoli.

After the stage of cheesy hepatization, whether lobular or miliary, is reached, four different events may follow. The exudation in the alveoli may take up sufficient serum to become fluid, be coughed out, and the lung resumes its normal character. Or, if the hepatization is of small amount, it remains quiescent, shrinks, becomes calcareous, and a fibrous capsule forms around it. Or, the exudation softens, and at the same time the walls of the alveoli, having lost their vitality, degenerate and disintegrate, and in this way are formed cavities with ragged walls and puriform contents. These cavities usually communicate with a bronchus, and sometimes merely enlarge a cavity already formed by a bronchiectasia. Or, the process of exudation into the air-cells may be accompanied by inflammation of the interstitial connective tissue and fibrous induration. When the disease is of long standing, this new formation of fibrous tissue

is the rule. We find the pleura thickened, bands and septa of fibrous tissue extend inward from the pleura into the lung tissue, the connective tissue around the vessels and bronchi is increased; around the various cheesy nodules there is a zone of new fibrous tissue, the walls of the alveoli hypertrophy, and so the process may go on until a large part of the lung may be converted into fibrous tissue and cheesy nodules. The new fibrous tissue may be black or slate-colored, or white or gray. In that which is formed around the bronchi and the vessels, and in the walls of the alveoli we can often see an early stage of the process, consisting in a development of great numbers of small round cells.

Fibrous Phthisis is usually associated with some degree of catarrhal phthisis, but may occur alone or form the principal part of the lesion. Like the catarrhal form, it may occur in small nodules, or be diffused over a large part of a lobe. The nodules are from the size of a pea to that of a walnut, of a dark slate-color, mottled with whitish points, of very hard consistence. They may be scattered in large numbers through both lungs, or there may be only two or three in all. They consist of pigmented fibrous tissue, with here and there groups of small, round cells, and sometimes the thickened walls of the alveoli can still be seen. The diffused form may involve part of a lobe or an entire lung. The pleura is thickened, and bands of white or slate-colored fibrous tissue extend inward. At the same time, the connective tissue around the bronchi and vessels and the walls of the alveoli increases until the pulmonary tissue is almost entirely lost in a fibrous mass. In this way, the apex of a lung, for example, may be changed into a firm mass traversed by dense bands of fibrous tissue, leaving a little lung tissue between them. This form of fibrous phthisis is usually associated with chronic bronchitis and bronchiectasia. The dilatation of the bronchi may be cylindrical or fusiform, or sacculated, or all these forms combined. The walls of the bronchi are frequently trabeculated. In this way, we may find an entire lung converted into a honeycomb of cavities, separated by thick bands of fibrous tissue.

Tubercular Phthisis is the form which is least frequent and

most difficult to study. It is seen in its most typical form in the lungs of children. Here we find both lungs studded with very small, grayish, semi-translucent granulations. When these granulations are discrete, and only their centres yellow and cheesy, they may readily be recognized. But when they become aggregated and entirely cheesy, it is difficult to distinguish them from the catarrhal miliary phthisis of children. In adults the same is the case. Occasionally we meet with pure and typical cases concerning which there is no doubt. The granulations are scattered in great numbers through both lungs. They are very minute, can hardly be felt on the surface of the section, are gray, semi-translucent; some have a white spot at the centre; the lung tissue between them appears normal. The granulations seem to grow from the walls of the bronchi and vessels, and sometimes from the walls of the air-cells. They consist of small, round, and fusiform cells, and sometimes of larger masses of finely granular matter, containing nuclei, (myéloplaxes.) The cells are considered by Virchow to be the same as the cells of lymphatic glands, and he calls the granulations heteroplastic lymphomata. Others regard the cells as belonging to connective tissue. The cells are contained in a matrix which is sometimes finely granular, sometimes a distinct fibrous network. There is almost always some catarrhal pneumonia of the adjoining lung tissue, though it may appear normal to the naked eye. There are usually small granulations of the same appearance in the serous membranes and in the liver, spleen, and kidneys.

This is the only form of tubercles in the adult lung, which can be certainly recognized. The small, whitish spots which are often seen in fibrous induration seem to be merely such accumulations of round cells as are to be seen in any new growth of fibrous tissue. There are, however, sometimes small opaque, cheesy, miliary nodules of which it cannot be certainly said whether they are tubercles or miliary hepatizations.

PULMONARY SYPHILIS.

There is much difficulty in determining what lesions of the lungs are dependent upon syphilis. It is generally admitted

that two lesions can be ascribed to this disease ; namely, Fibrous induration, and Gummy tumors.

The fibrous induration may occur in the form of multiple, hard, slate-colored, or white nodules, or of a diffused induration of part of a lobe. It does not differ anatomically from the lesions of fibrous phthisis, and is only to be diagnosticated with the concurrence of other syphilitic lesions and history.

The gummy tumors are found in small numbers, from one to six or eight. They form nodules of the size of a pea to a walnut, partly white, partly gray-colored, imbedded in fibrous induration. They are cheesy in irregular patches, separated by bands of fibrous tissue, and consist of small, round, and fusiform cells. As many cheesy nodules of other origin are found in the lungs, gummy tumors are only to be pronounced to be such after very careful examination.

In still-born children, parts of the lungs are sometimes found in a condition of white hepatization, the alveoli being filled with great numbers of epithelial cells. This lesion is usually attributed to syphilis.

NEW GROWTHS.

Cysts are of very rare occurrence in the lungs. They may contain serum, fat, cholesterine, and hair.

Fibroma is also very rare. Rokitansky describes the tumors as small, white, and firm ; or yellow, flabby, and puckered.

Enchondroma may be primary or secondary. The primary tumors are small, multiple, usually at the root of the lung, often attached to the bronchi and vessels. They are sometimes found after death, when they had given rise to no symptoms during life. The secondary tumors are more common. They may reach a large size, sometimes occupying an entire lung.

Osteoma is rare. It forms either irregular plates, or somewhat globular, spongy tumors. It is not to be confounded with calcification of the lung tissue.

Sarcoma is not infrequent. It occurs in the form of rounded nodules, usually multiple. It is secondary to sarcomata in other organs, especially to those connected with the bones.

These tumors have the same structure as the primary tumors, but may be pigmented when the latter are not.

Carcinoma is usually secondary, but may be primary. It may be medullary, melanotic, epithelial, or colloid. It occurs as nodules of various size and number, or may infiltrate large portions of the lung. It may spread to the pleura, the pericardium, the heart, and the walls of the thorax, and appear on the outside of the chest.

Pigment is found regularly to a considerable amount in the adult lung. In old age, the quantity may increase very considerably. It is a very common thing to find, under the pleura, little nodules composed entirely of pigment. Fibrous induration, and the pigment induration of heart-disease, are accompanied by an increased formation of pigment. In miners, charcoal-workers, grinders, and persons who constantly inhale foreign particles, the lungs become intensely pigmented—anthracosis. In them the pigment is partly the foreign particles themselves, and partly produced by the chronic bronchitis. In all the cases, the pigment is found in the connective tissue and epithelial cells, in the form of granules or masses. It is probably derived from the coloring matter of the blood, in consequence of an abnormal stagnation in the capillaries.

Myoma I have seen in one instance. It occurred in a man aged 52, who had symptoms of bronchitis and laryngitis. The tumors formed large white nodules in the upper lobe of the lung. The nodules consisted almost entirely of smooth muscle.

PARASITES.

Echinococci occur in the lungs as a cyst, surrounded by a thick fibrous capsule. The capsule may suppurate and the sac be emptied into a bronchus, into the pleura, through the chest-wall, through the diaphragm, or into the intestines.

Cysticercus Cellulosæ is not frequent in the lungs.

Strongylus Longevaginat has been seen in a child's lung.

In gangrenous cavities of the lungs, vegetable parasites, belonging to the varieties aspergillus, mucor, and sarcina, have been observed.

THE PLEURA.

HYDROTHORAX.

Non-inflammatory exudations of serum into the pleural cavities accompany the various diseases which cause general hydræmia. A small amount of serum, either clear or bloody, is often found as a mere post-mortem change.

All chronic and exhausting diseases, renal disease, cardiac disease, and cirrhosis, are very often attended with hydrothorax. The fluid is a clear serum, sometimes yellow, which may reach such an amount as to compress the greater part of the lung.

HÆMORRHAGE.

Small, dark-red extravasations in the pleura are found with purpura, death by suffocation, and in the lungs of young children. Hæmorrhages into the cavity of the pleura are caused by the rupture of aneurisms, by the rupture of the pericardium previously distended with blood, and by wounds of the chest-wall.

INFLAMMATION.

Pleurisy occurs as a primary disease, or from inflammation of the lung tissue, or from perforation of the chest by wounds, by abscesses of the wall of the thorax, of the liver, spleen, kidneys, and around carious vertebræ. It occurs also without perforation with perihepatitis and perisplenitis. Pyæmia and puerperal fever also often cause pleurisy.

Inflammation of the pleura is attended with exudation of serum, fibrine, lymphoid cells, and sometimes with extravasation of blood. The amount of these different substances varies in different cases.

(1.) There may be a scanty exudation of fibrine alone. The surface of the pleura is found coated with a thin, membranous, grayish layer, consisting of fibrine entangling a few lymphoid cells. This may be entirely absorbed, or it may cause permanent adhesions between pulmonary and costal pleura, or between the different lobes. The adhesions are most frequently

found after a number of such attacks. This form of pleurisy usually gives no marked symptoms during life, and is frequently overlooked. It is very common to find, at the post-mortem, old adhesions, the existence of which was not suspected during life.

(2.) The exudation consists of fibrine and serum. The pleural cavity is partly or completely filled with turbid serum, mixed with flocculi of fibrine, and a few lymphoid cells. The pleura itself is coated with a yellow, membranous layer, composed of fibrine and lymphoid cells. Sometimes there are extravasations of blood on the surface of the pleura, and in the fluid. The serum usually occupies the lower portion of the pleural cavity, but sometimes is confined by adhesions and false membranes to other portions of the cavity. If the serum occupies the lower half of the cavity, the lower lobe of the lung is compressed and unaerated, and the entire lung pushed upward. If the cavity is full of serum, the entire lung is compressed toward its root, and against the vertebral column. Such a pleurisy, with effusion, may entirely disappear, leaving only some adhesions behind it; or it may continue so long without absorption of the fluid that the patient dies from its effect, or the effusion becomes purulent. If absorption takes place, the lung tissue resumes its normal consistence, although sometimes, when the effusion is of very long standing, the lung may remain compressed and bound down by adhesions.

(3.) There is an exudation of purulent serum. The pleural cavity is found filled with yellow, often stinking fluid, the pleura is coated with fibrine and pus. The fluid is usually in large quantities, and in the lower part of the pleural cavity. Sometimes, however, it occurs in the form of circumscribed collections of pus between the pulmonary and costal, or diaphragmatic pleura, entirely shut in by adhesions. Such a purulent pleurisy, or empyema, is either fatal, or may be absorbed, or may perforate the chest-wall and escape externally, or may perforate the lung and escape through a bronchus, or may perforate the diaphragm, and find its way downward behind the peritoneum. Wunderlich describes a suppurative peri-pleuritis which forms abscesses between the costal and dia-

phragmatic pleura and the chest-wall, and between the pulmonary pleura and lung.

In pleurisy of long standing, the thickening and adhesions of the pleura are prominent lesions. In fibrous phthisis and in chronic catarrhal phthisis, the pulmonary pleura is often enormously thickened. The pulmonary and costal pleura may become inseparably adherent over an entire lung, or they may be united at various points by firm membranous adhesions. These adhesions and thickenings may, after a time, become calcified.

HYDROPNEUMOTHORAX.

The pleural cavity may contain air. The air enters either through a perforation of the chest-wall or of the lung.

The air is usually accompanied by a purulent exudation in the pleural cavity.

In phthisis, in pyæmic infarctions, gangrene, hydatid sacs, empyema, emphysema, bronchiectasia, abscesses of the adjoining soft parts, perforation of the œsophagus, and in wounds of the thorax, air may enter the pleural cavity.

NEW GROWTHS.

Fibroma occurs in the form of small, white nodules; of poly-poid tumors, as large as a walnut; and of small nodules, free in the pleural cavity.

Lipoma occurs in the form of large masses growing from the connective tissue beneath the costal pleura and pushing this membrane inward.

Angioma has been seen in the costal pleura.

Cysts, both serous and dermoid, are sometimes found.

Tubercles are found in the pleura as small, gray, miliary granulations. They are often found with tubercular and catarrhal phthisis.

Carcinoma is secondary to that disease in other organs, especially in the mamma. It forms small, whitish nodules in the costal and pulmonary pleura. They are either soft or hard. With cancer of the mediastinum the pleura is often involved. I have seen a case in which there was a layer of new growth, two inches thick, on the outer surface of the costal pleura.

The pleural cavity remained open, and was filled with serum containing fibrine and blood.

Leukemic Tumors occur as soft, white, flat, or rounded masses, composed of cells like those in the lymphatic glands.

PARASITES.

Echinococci are found in the costal and pulmonary pleura and in the anterior mediastinum. Sometimes they perforate from the liver into the pleural cavity.

THE MEDIASTINUM.

INFLAMMATION.

Abscesses are found in the anterior and posterior mediastinum. They are caused by fractures, caries, or necrosis of the sternum and vertebræ, by perforation of the œsophagus, by suppuration of the lymphatic glands, or may be idiopathic. The pus is formed slowly, and may accumulate in large quantities. The heart and lungs may be displaced and the sternum bulged outward. They may cause pleurisy or pericarditis, or may perforate outward, or into the pleural cavity, the œsophagus, the trachea, or a bronchus.

NEW GROWTHS.

Lympho-Sarcoma occurs in the form of large lobulated tumors, commencing in the lymphatic glands. The cervical, bronchial, and mediastinal glands are most frequently involved, and form lobulated tumors, which may occupy a large part of the cavity of the thorax. A persistent thymus gland may also be the point of origin. These tumors at first retain the character of enlarged glands inclosed in a capsule; later, the new growth invades and breaks through the capsule, and infiltrates the neighboring soft parts. Later still, metastatic tumors are formed in the lungs, liver, spleen, and kidneys. The lymphatic glands in other parts of the body may be enlarged at the same time. The growth of these tumors is rapid, and they soon prove fatal.

Sarcoma and Carcinoma grow as primary tumors within the mediastinum. They originate in the periosteum of the sternum, in the outer layers of the parietal pericardium, and in the connective tissue of the mediastinum. They grow very rapidly, fill up a large part of the thorax, compress and invade the neighboring soft parts, and form metastatic tumors in other organs.

Teratoma myomatodes.—Under this name Virchow describes a very remarkable tumor. It grew rapidly in a man, aged 22, of good constitution and physical development. The right pleural cavity was nearly filled with a solid growth. There were similar growths, of smaller size, on the left rib, in the liver, spleen, and kidneys.

The mediastinal tumor consisted of two portions, a larger and a smaller.

The larger portion consisted of solid tissue, in which were a few cavities. It was very vascular. Part of it was composed of dense fibrous tissue. The greater portion, however, consisted of loose connective tissue fibres, of fusiform cells, and of large, many-nucleated cells. Some of the fusiform cells were small, and of the usual appearance; but many were large, and presented striations like those of young voluntary muscle.

The smaller portion of the tumor looked like a multilocular cystoid. The cysts varied much in size, and contained serum, colloid matter, and blood. Some of the cysts contained a thick, white material, in which were hairs, epidermic scales, ciliated epithelium, and cholesterine. In the fibrous tissue about these cysts were pieces of hyaline cartilage. Near the surface of the tumor the tissue consisted of a fibro-cellular stroma forming alveoli filled with epithelium.

The tumor on the third left rib consisted of connective tissue fibres and cells, muscle cells, cysts, alveoli filled with epithelium, and a small portion of tissue resembling foetal lung.

THE VASCULAR SYSTEM.

THE PERICARDIUM.

MALFORMATIONS.

THE pericardium may be absent, when there is ectopia of the heart. Cases have been observed in which the heart and the lung were situated in the left pleural cavity, with only rudiments of the parietal pericardium at the base of the heart.

INJURIES.

The pericardium may be wounded by penetrating weapons, by gunshot wounds, and by fragments of bone.

It may be ruptured by severe contusions of the thorax, and by rapid extravasation of blood into the pericardial sac.

Perforations may be produced by empyema, by mediastinal abscesses, by abscesses of the chest-wall and of the liver, by aneurisms of the aorta, and by suppurative inflammation of the pericardium.

DROPSY.

In most post-mortems, we find a little serum, from $\frac{1}{2}$ ounce to 1 ounce, in the pericardial sac. This serum is usually clear and of a light yellow color; if decomposition has commenced, it may be of a reddish color, or it may be slightly turbid from the falling off of the pericardial epithelium.

Large accumulations of serum are found as part of general dropsy from heart-disease, kidney-disease, etc. The serum is clear and of a light yellow color. Hydro-pericardium is usually moderate in comparison with the accumulations of serum in the other serous cavities; sometimes, however, there is a very large

amount of serum, which hinders the movements and the nourishment of the heart.

HEMORRHAGE.

Extravasations of blood in the cavity of the pericardium are produced by wounds and rupture of the heart, rupture of the aorta and of aneurisms, and occur with pericarditis. Small extravasations in the substance of the pericardium are found with scurvy, purpura, fevers, etc.

PNEUMATOSIS.

Air in the pericardium is sometimes found as a post-mortem appearance, accompanied with drying of portions of the pericardium.

Wounds or paracentesis of the pericardium; the perforation of ulcers of the stomach, cavities of the lungs, and ulcers of the œsophagus may admit air into the pericardial cavity. Pericarditis, with foul, purulent exudation, may evolve gases.

INFLAMMATION.

Pericarditis.—The earliest change in pericarditis is a congestion of the pericardium; this, however, we very frequently fail to find at the post-mortem. Small ecchymoses are not uncommon. The inflammation is attended with the production of serum, fibrine, pus, and blood in variable quantities. In very mild cases, there are only one or two ounces of turbid serum in the pericardium. In severer cases, the serum reaches the amount of several ounces, or even pounds, accumulating first at the base of the heart, and afterward surrounding it more uniformly.

Instead of turbid serum there may be great numbers of pus globules, so that the pericarditis is suppurative. In rare cases, the pus may become foul and gangrenous.

In other cases, there is but little serum or pus; but the pericardium is coated with fibrine in a thin layer, or in large quantities, sticking the visceral and parietal pericardium loosely together.

With the serum, the pus, or the fibrine, there may be an extravasation of blood, sometimes in large amount.

Pericarditis may run an acute or a chronic course. It may end in death; in absorption of the serum; in membranous thickenings of the pericardium; in adhesions between its visceral and parietal portions; or the pus and fibrine together may undergo cheesy degeneration.

With large, serous effusions, especially when they are chronic, the heart-wall becomes soft and flabby, and there may be consequent dilatation of the ventricles. There may be also granular degeneration of the muscular fibres, especially of those near the pericardium.

In some cases of chronic pericarditis we find irregular calcific plates. The calcification takes place in the membranous thickenings and adhesions of the pericardium, in the parietal pericardium, or in the cheesy masses of thickened pus which are found with and in extensive adhesions.

NEW GROWTHS.

Carcinoma is found in the pericardium in the form of small nodules, metastatic tumors from cancerous or epithelial growths of other parts. In cancers of the mediastinum, the greater part of the parietal pericardium may be involved in the new growth.

Sarcoma is found in the same way in metastatic nodules, and as a continuous infection from mediastinal sarcomata.

Fibroma occurs rarely. It may have the polypoid form, and the pedicle may atrophy so as to leave free fibrous bodies in the pericardial cavity.

Cysts of the visceral pericardium have been described by Rokitansky.

Tubercles in the miliary form are found in the pericardium, and in inflammatory membranes.

THE HEART.

CONGENITAL MALFORMATIONS.

The malformations of the heart are usually closely connected with malformations of the aorta and pulmonary artery. They depend on arrest of, or abnormal development; on endocarditis, myocarditis, thrombosis, or mechanical causes.

I. The common arterial trunk is only partially, or not at all, separated into aorta and pulmonary artery. The divisions between the heart cavities are at the same time defective.

(1.) There is one ventricle and no auricle.

(2.) There is one ventricle and one auricle.

(3.) There is one ventricle and two auricles; the aorta is alone or incompletely separated from the pulmonary artery.

II. The trunk of the pulmonary artery, or of the aorta, is stenosed or obliterated, and from the obstruction to the current of blood the development of the septa, between the heart cavities, is prevented.

(1.) The aorta, at its origin or in the ascending portion of the arch, is stenosed or closed. The pulmonary artery gives off the descending aorta, and supplies the carotids and subclavians. The foramen oval remains open, or there is no septum between the auricles. The ventricular septum is also usually defective. The right ventricle is hypertrophied.

(2.) The pulmonary artery is stenosed or closed. Its branches are supplied by the aorta, through the ductus arteriosus. The ventricular septum is defective, the foramen ovale is open, or the auricular septum defective.

III. The malformation affects the aorta and pulmonary artery after they are more fully developed.

(1.) There is stenosis of the aorta between the left subclavian and ductus arteriosus, or just at the opening of the ductus arteriosus. The descending aorta is then a continuation of the pulmonary artery.

(2.) The aorta gives off all its branches from the arch, but the descending aorta is a continuation of the pulmonary artery, or the carotids may spring from the aorta, the subclavians from the pulmonary artery.

(3.) The vessels are transposed, the pulmonary artery arises from the left, the aorta from the right ventricle; the pulmonary veins empty into the left, the venæ cavæ into the right auricle; or the veins also may be transposed. The septa are defective.

IV. The aorta and pulmonary artery are normal, but the cardiac septa are defective.

(1.) The foramen ovale remains partly open. This condition may continue through life without giving any trouble.

(2.) The ductus arteriosus may remain open for many years; this, also, causes no disturbance.

(3.) There is a small or large opening in the ventricular septum. This may give rise to no symptoms unless disease of the heart or lungs be superadded.

V. Either of the auriculo-ventricular orifices may be entirely closed. The foramen ovale remains open, and the ventricular septum is defective.

VI. The valves of the different orifices of the heart may be absent or defective. The arteries or the ventricles are usually defective at the same time.

The aortic and pulmonary valves may consist of two large or four small leaves, instead of the usual three.

Generally speaking, the existence of openings between the two auricles or the two ventricles, admitting some admixture of venous and arterial blood, produces no change in the circulation. If, however, the passage of the current of venous blood into the right heart is in any way interfered with, the consequences are very serious. Cyanosis is produced, the skin is of a bluish color, the small veins and capillaries are dilated, exudation of serum and hypertrophy of connective tissue takes place, especially in the fingers and toes.

Besides the malformations already mentioned, we may find :

Entire absence of the heart.

Abnormal septa and chordæ tendineæ in the heart cavities.

Abnormal shapes of the heart.

Abnormal positions of the heart.

(a.) There is a smaller or larger defect in the walls of the thorax, so that the heart projects on the outside of the chest; the pericardium is usually absent.

(b.) The diaphragm is absent, and the heart is found in the abdominal cavity.

(c.) The heart is placed in some part of the neck or head; this only occurs in fœtuses very much malformed.

(d.) The heart is transposed, being on the right side.

Very rarely two more or less perfect hearts are found in the same thorax.

HYPERTROPHY.

All the cavities of the heart may have their walls hypertrophied, or the thickening may only involve one or more.

While the wall of a ventricle is thickened, its cavity may retain its normal size, or be dilated or contracted. The existence of the last condition—concentric hypertrophy—is denied by some authors, affirmed by others.

Hypertrophy of both ventricles increases both the length and breadth of the heart. Hypertrophy of the left ventricle (alone) increases its length. The apex is then lower and further to the left than usual. Hypertrophy of the right ventricle (alone) increases the breadth of the heart toward the right side; but sometimes the right edge of the heart retains its normal situation and the apex is displaced to the left. With large hypertrophy of both ventricles, the base of the heart may sink so that its long axis approaches a horizontal direction.

The exact way in which hypertrophy of the heart-walls takes place, whether by increase in the size or in the number of the muscular fibrillæ, or in both ways, is not definitely determined.

Hypertrophy of the heart may depend upon a variety of causes.

(1.) Changes in the valves, either insufficiency or stenosis, produce hypertrophy in the cavities to which the valves belong.

(2.) Obstruction to the passage of blood through the aortic system of vessels, by aneurisms, dilatation, atheroma, congenital stenosis, the pressure of tumors, disease of the kidneys, (especially atrophy,) leads to hypertrophy, first of the left ventricle, and secondarily of the right ventricle.

(3.) Obstruction to the passage of blood through the pulmonary artery, by stenosis, and by diseases of the lungs, produces

hypertrophy of the right ventricle, and secondarily of the right auricle and left ventricle.

(4.) Any cause, whether muscular or nervous, which increases the rapidity and force of the heart's contractions, may produce hypertrophy.

(5.) Dilatation of the ventricles from any cause is frequently followed by hypertrophy.

(6.) Pericarditis may produce hypertrophy by inducing softening and dilatation of the ventricles, or by leaving adhesions which obstruct the heart's action.

Finally, for some cases of hypertrophy no satisfactory cause can be found.

DILATATION.

Dilatation may be active—combined with hypertrophy; simple—the heart-walls retaining their normal thickness; or passive—with thinning of the walls.

Either one or all of the heart cavities may be dilated. The auricles are most frequently affected; next, the right ventricle; least often, the left ventricle.

Active dilatation has already been described under the head of hypertrophy.

In simple dilatation, the shape of the heart is changed according to the cavities affected. Dilatation of the right ventricle increases the breadth, dilatation of the left ventricle, the length of the heart. The walls of the heart are of normal thickness; the muscular tissue is flabby; the muscular fibres are normal, or fatty, or waxy.

Passive dilatation is produced by:

(1.) Changes in the valves. Mitral or aortic stenosis, or insufficiency, may produce dilatation of the auricles and right ventricle. Pulmonary stenosis, or insufficiency, may produce dilatation of the right auricle and right ventricle. Aortic insufficiency, with or without stenosis, or mitral insufficiency, may produce dilatation of the left ventricle. Dilatations from these causes are often succeeded and compensated for by hypertrophy of the heart's walls.

(2.) Changes in the muscular tissue of the heart-walls.

Serous infiltration of the heart-wall, from pericarditis ; myocarditis ; fatty degeneration of the muscular fibres ; and atrophy of the muscle, from chronic diseases, may all lead to dilatation.

(3.) A heart which is already hypertrophied may, from degeneration of the heart-wall, become dilated.

(4.) Acute infiltrations of the lungs, and acute pleuritic exudations, by rendering a large number of vessels suddenly impermeable to the blood current, produce a rapid stasis in the pulmonary artery and dilatation of the right heart.

ATROPHY.

Atrophy of the walls of the heart may be accompanied with no change in the size of its cavities ; or with dilatation, (the same as passive dilatation ;) or, more frequently, with diminution in the size of the cavities.

The atrophy involves most frequently all the cavities of the heart, but may be confined to one or more of them.

The muscular tissue appears normal, or brown from the presence of little granules of pigment in the muscular fibres ; or the muscular fibres may undergo fatty degeneration ; or there may be an abnormal accumulation of fat beneath the pericardium ; or there may be a peculiar gelatinous material beneath the pericardium ; this consists of fat which has undergone mucous degeneration.

The causes of atrophy of the heart are :

(1.) It is a congenital malformation ; the heart of an adult then looks like that of an infant.

(2.) Any chronic and exhausting disease, repeated hæmorrhages, old age, typhus fever, dysentery, etc., may produce atrophy.

(3.) Chronic pericarditis, with large serous effusion, or with thickening of the epicardium, producing constriction of the coronary arteries.

(4.) Stenosis, atheroma, calcification, or thrombosis of the coronary arteries, may produce partial or total atrophy.

(5.) Myocarditis, with fatty or fibrous degeneration.

(6.) Mitral stenosis may cause atrophy of the left ventricle.

CHANGES IN POSITION.

Changes in the position of the heart are congenital or acquired. The congenital malpositions have been already noticed ; the most important clinically is the transposition of the organ to the right side.

The acquired malpositions are caused by :

(1.) Hypertrophy of the heart ; its long axis approaches the horizontal direction.

(2.) Changes in the thoracic viscera. Emphysema of both lungs pushes the heart downward. Emphysema, pleurisy, with effusion, or pneumo-thorax of one side, push the heart to the other side. Pleurisy or chronic pneumonia, producing retraction of one side of the thorax, draws the heart to that side. New growths, aneurisms, and curvatures of the spine displace the heart in various directions.

(3.) Changes in the abdomen. Accumulations of fluid and new growths in the abdomen, and tympanites, may push the heart upward.

WOUNDS AND RUPTURES.

Wounds of the heart are produced by penetrating instruments, by bullets, and by fragments of bone. The right ventricle is the more frequently wounded ; next, the left ; rarely, the auricles.

The wound may penetrate into the cavities of the heart, or only pass partly through its wall, or a bullet, or the broken end of a weapon, may be imbedded in the wall.

If the wound penetrates into a cavity, and is gaping, death follows instantly, and the pericardium is found filled with blood. If the wound be small and oblique, the blood may escape gradually, and death does not ensue for several days. In rare cases, adhesions are formed with the pericardium, and the wound cicatrizes.

Wounds which do not penetrate may cause death by the inflammation which they excite, or may cicatrize.

Bullets and foreign bodies may become encapsuled in the heart-wall, and remain so for years.

Ruptures of the heart-wall occur in various ways.

(1.) Severe contusions of the thorax may produce rupture, usually of one of the auricles.

(2.) Spontaneous rupture occurs usually in advanced life. The left ventricle is the cavity most frequently ruptured. There is usually one rupture, sometimes two; five have been seen. The rupture is usually oblique, larger internally than externally; there may even be a small cavity filled with blood in the heart-wall. The heart-wall may be of normal thickness, or thin; it is soft and friable, the muscular fibres advanced in fatty degeneration. The rupture usually takes place while the patient is quiet. Death is usually instantaneous, but sometimes does not ensue for several hours.

Rupture of the inner layers only of the heart-wall may give rise to aneurisms of the heart.

In rare cases, circumscribed inflammation, abscesses, hydatids, and tumors may produce rupture.

(3.) In very rare cases, rupture is produced by stenosis of the aorta and dilatation of the heart cavities.

(4.) Rupture of the papillary muscles and tendons is produced by fatty and atheromatous processes.

INFLAMMATION.

MYOCARDITIS.

Our knowledge of inflammation of the walls of the heart is still imperfect. The distinction between parenchymatous degeneration, and parenchymatous inflammation and their relations to changes in the interstitial connective tissue, are obscure. In chronic pericarditis with effusion, we may find in the outer layers of muscle marked granular degeneration of the muscular fibrillæ. This may be looked on as a parenchymatous inflammation, due to irritation, or as a simple degeneration from impaired nutrition.

A general and acute inflammation of the entire heart is described by Rindfleisch. It occurred in a man with a syphilitic history, and caused sudden death. The heart-walls were firm and stiff, the muscular tissue was of a fresh violet red color, iridescent, semi-translucent, of the consistence of caoutchouc.

The muscular fibres contained a finely granular substance collected around the nuclei, they were broken into quadrangular fragments.

Abscesses of the heart-wall may be due to embolism or to unknown causes. Those due to embolism are multiple, scattered over the surface of both ventricles, and have the ordinary characteristics of pyæmic abscesses.

Those due to other causes are usually single, situated in the wall of the left ventricle, or in the septum, and may be as large as a walnut. The abscess contains pus and broken-down muscular fibres. The pus may become thickened, cheesy, and inclosed in a fibrous capsule. More frequently the abscess opens into the pericardium or into the heart-cavity. If it opens into a heart-cavity, the blood enters and becomes mixed with the contents of the abscess, and a very favorable condition is afforded for the production of emboli in different parts of the body. The blood may finally rupture into the pericardial cavity, and cause death. If the abscess is in the interventricular septum, it may establish an opening between the two ventricles, or it may extend upward and form a diffused abscess in the connective tissue at the base of the heart.

Change of the muscular substance of the heart into fibrous tissue is sometimes found. It usually occurs in the left ventricle, and in connection with endocarditis. We find a circumscribed portion of the heart-wall converted into fibrous tissue of some thickness, or into a thin membrane. It seems natural to ascribe such a condition to chronic inflammation going on in the heart-wall.

These cicatricial spots may lead to the formation of aneurisms of the heart, or to general dilatation.

THE ENDOCARDIUM.

The endocardium resembles the entire coat of a blood-vessel spread out over the inner surface of the heart. It consists of several layers. On its inner surface is a layer of pavement endothelium. The cells are placed on a thick lamella, formed of anastomosing elastic fibres; beneath this is a layer of loose elastic fibres mixed with a little connective tissue and some smooth

muscular fibres. A layer of connective tissue separates the endocardium from the muscular tissue of the heart. The valves are formed by a duplicature of the endocardium, so that there is on each face of the valve a layer of epithelium, an elastic layer, and in the centre a thin layer of connective tissue. Blood and lymph vessels are found in the endocardium.

In intra-uterine life, endocarditis usually attacks the right ventricle; after birth, the left ventricle. The valves are the portion of the endocardium which is most frequently diseased.

Rheumatism, Bright's Disease, and the severe febrile diseases are considered the usual causes of endocarditis, but there are many cases for which no cause can be found.

The most moderate degree of inflammation manifests itself in a simple swelling of the valves. The valves are thickened and feel succulent and elastic. The swelling seems to be due to a simple swelling of the anatomical elements, with some increase in number of their connective tissue-cells.

Another mild form exhibits itself in a simple opacity and thickening of the endocardium. This is usually seen in the left ventricle, in its upper part on the posterior wall; it may or may not be associated with disease of the valves.

In more intense inflammation, the increase of cells is more evident, so that we see little granulations on the valves or other parts of the endocardium. The cells increase in number till they reach the inner surface of the endocardium, and break through in a little fungous mass. On this roughened surface fibrine from the blood in the heart may be deposited so as to form vegetations of considerable size. Sometimes the cell production may be so great that minute abscesses are formed in and beneath the endocardium.

Much more common than all of these changes is chronic endocarditis, or atheroma, as it is more often called. The process consists in a production of small round cells, in the substance of the endocardium, followed by granular, fatty, or calcific degeneration. So that we find in the endocardium patches of an opaque, cheesy substance, made up of granules and debris of cells; or calcareous plates. Over these patches, the inner layer of the endocardium may be destroyed and the patches may re-

main exposed, or the cheesy matter may soften and give rise to ulcerative destruction of tissue.

In this way we find a great variety of lesions produced in the valves, and less frequently in the endocardium of the ventricles. The valves are simply thickened by cheesy yellow patches; or they are stiff and rigid from cheesy or calcareous plates; or they are drawn together so as to obstruct the openings to which they belong; or the atheromatous matter softens, breaks down, and a larger or smaller part of the valve is destroyed. As soon as a roughened surface is produced, fibrine is usually deposited on it in the form of vegetations.

The endocardium of the ventricles presents the same lesions: cheesy and calcareous plates and ulcerated surfaces. In the latter case, the muscular tissue may become involved and excavations of some little depth produced. In some cases, the papillary muscles or chordæ tendineæ are destroyed so as to leave the valves flapping.

CHANGES IN THE VALVES.

Fenestration of the valves is usually a change productive of no bad consequences. It occurs in the aortic and pulmonary valves. The valves are thinner than usual, and close to their free edges are small slits extending from the centre to the attached edges of a leaf.

Aneurisms of the valves are produced in two ways.

(1.) They are the result of endocarditis. One of the lamellæ of the leaf of a valve is destroyed and the other lamella is converted into a sac filled with blood. These aneurisms are found in the aortic valve, projecting into the ventricle; and in the mitral valve, projecting into the auricle. Not infrequently the wall of the aneurism gives way, so that there is a rupture entirely through the valve.

(2.) The entire thickness of a leaf of a valve is converted into a sac filled with blood. This occurs in the aortic, mitral, and tricuspid valves; its cause is unknown.

Hæmorrhage in the substance of the valves is sometimes found in very young children. It does not appear to have much clinical importance.

Insufficiency of the valves to close the orifice to which they belong is produced by :

(1.) Inflammation, thickening, shrinking, and shortening of the valves. To these may be joined atheroma, calcification, and vegetations.

(2.) The valves may become adherent to each other or to the heart-wall.

(3.) Partial destruction of a valve, of a papillary muscle, or of one of the chordæ tendineæ by inflammation.

(4.) Dilatation of the orifice of a valve without corresponding increase in the size of the valve itself.

(5.) A very high degree of fenestration of the valve.

(6.) Large vegetations adhering to the valve.

Stenosis of the valves is produced by inflammatory changes resulting either in simple rigidity, or in adhesions and contraction of the valve. In the latter case, the stenosis may be so marked that a quill will hardly pass through the orifice.

The same lesions frequently produce both stenosis and insufficiency of a valve.

These changes occur most frequently in the aortic and mitral valves; more rarely, in the pulmonary and tricuspid.

Insufficiency and stenosis of the valves produce changes in the heart itself and in the rest of the body.

In the heart we find dilatation and hypertrophy in various degrees.

Insufficiency of the mitral valve produces dilatation and hypertrophy of the left ventricle, dilatation of the left auricle, and not infrequently dilatation and hypertrophy of the right ventricle and auricle.

Stenosis of the mitral valve usually leaves the left ventricle of normal size, or even atrophied; the left auricle, right ventricle, and right auricle are usually dilated and hypertrophied.

The tricuspid valve may be insufficient or stenosed, usually both at once. A small degree of insufficiency appears to be a physiological condition. Greater degrees are usually secondary to mitral stenosis. If stenosis exists, it is moderate in degree, and its effect is the same as insufficiency—dilatation of the right auricle and of the veins leading into it.

The aortic valve may be insufficient, or stenosed, or both.

Insufficiency produces dilatation or hypertrophy of the left ventricle, secondarily of the left auricle, right ventricle and auricle. Stenosis, with insufficiency, produces the same lesions in a higher degree. Stenosis alone produces dilatation of the left ventricle without hypertrophy, giving the heart a globular form.

The pulmonary valve is rarely insufficient or stenosed. It causes dilatation or hypertrophy of the right ventricle and auricle.

The changes produced by valvular lesions in other parts of the body are due to an increase of venous, or a diminution of arterial pressure, both resulting in the same way, namely, in a constant venous congestion of the different viscera, and serous effusions into the different serous cavities. Stenosis of the mitral valve, or of the pulmonary artery, produces these changes in the highest degree.

Pigment induration of the lungs, congestion and subsequent changes in the liver, catarrhal inflammation of the stomach and intestines, induration of the kidneys, and cyanosis of the skin may all be found.

ANEURISM OF THE HEART.

Sacs filled with blood, situated in the walls of the heart, and communicating with its cavities, are formed in several different ways.

(1.) In consequence of inflammatory processes in the endocardium and muscular tissue, a small or large portion of the wall is converted into fibrous tissue. The portion thus changed no longer resists the pressure of the blood from within, and is driven outward. Such a pouch may be a circumscribed sac, communicating with the heart-cavity by a small opening, or may look like a dilatation of part of the ventricle. The wall of such an aneurism becomes thinner as the sac increases in size. It is composed of the endocardium, new fibrous tissue, visceral pericardium, and sometimes the adherent parietal pericardium. The walls may calcify; or rarely they become so thin as to rup-

ture externally or into the right ventricle. The sacs may contain fluid blood, or be filled up with fibrine.

Such aneurisms are usually situated in the wall of the left ventricle; rarely in that of the left auricle. If they are in the septum, they may project into the right ventricle. They are usually single; sometimes two or three are found in the same heart.

(2.) Fatty degeneration of the heart-wall may reach such a point that the wall yields and is pouched out into an aneurismal sac.

(3.) Endocarditis and myocarditis, or fatty degeneration, may so soften a portion of the heart-wall that the endocardium and part of the muscular tissue is ruptured, and a ragged cavity is formed. This form of aneurism usually does not attain a large size, but soon ruptures externally, and causes the death of the patient.

THROMBOSIS OF THE HEART.

It is very common to find after death, in the heart-cavities, yellow, succulent, semi-translucent masses. They are most common and of firmest texture in persons who die of acute inflammatory diseases. They may adhere quite firmly to the walls of the heart, and may extend in long, branching cords into the vessels. They are formed in the last hours of life, and just after death. They have no clinical or pathological importance.

Coagulations of the fibrine of the blood in the heart do, however, occur during life, and may exist for years. If the fibrine adheres to the valves in small masses, these are called vegetations; if it coagulates in the heart-cavities in larger bodies, they are called thrombi, or heart polypi.

Such thrombi are found in all the heart-cavities. They form flattened masses firmly adherent to the endocardium; or rounded bodies in the spaces between the trabeculæ; or have a poly-poid shape and are attached by a narrow pedicle, or are free in the cavity.

They are usually found in connection with some valvular

lesion, which prevents the free circulation of blood through the heart.

They are firm, dry, and of a whitish color, they may soften and break down at their centres, so as to look like cysts filled with pus, or they may calcify. They are usually entirely unorganized, consisting simply of fibrine.

I have observed one case in which the thrombi were distinctly organized. The patient was a man found dead in the street, concerning whom no history was obtained.

There were two thrombi attached to the endocardium of the right auricle at a point half an inch from the tricuspid valve, and close to each other. One of them was spherical, $2\frac{1}{2}$ inches in diameter, attached to the auricle by a small, round pedicle. The other was ovoid, $3\frac{1}{2}$ inches long, was attached by a small pedicle, projected through the tricuspid valve into the right ventricle, was calcified, and its free end ragged and broken. When they were fresh, both polypi were formed of a yellow, semi-translucent, elastic tissue, parts of which were calcified. Small vessels filled with blood could be seen near the surface. A thin membrane continuous with the endocardium could be dissected up from their surfaces. The body of the polypi, where they were not calcified, consisted of a basement substance partly fibrous, partly homogeneous, and of round, oval, fusiform, and stellate cells. Arteries and capillaries, some containing blood, were found in considerable numbers.

I have also seen another organized thrombus in the right auricle. The specimens were shown to me by Dr. Mann.

The patient was a middle aged-man, one of whose legs was amputated on account of some tumor. After a time, the tumor recurred in the stump. At the autopsy there were found secondary nodules in the lungs, and a large thrombus in the right auricle and extending into the inferior vena cava. The growth in the stump, in the lungs, and in the auricle, all had the same structure. They were composed of a fibrous stroma inclosing rounded cavities. These cavities were filled with large nucleated cells resembling pavement epithelium. The cells were not in contact with each other, but were separated by a fine fibrous reticulum.

DEGENERATIONS.

Fatty Degeneration affects the heart in two ways, as an increase of the subpericardial fat, and as a change in the muscular fibres.

The increase of the subpericardial fat may be attended with atrophy of the muscular tissue, or it may not. It accompanies an increase of adipose tissue throughout other regions of the body.

Fatty degeneration of the muscular fibres produces fine granules in them, which may be so numerous as to apparently fill the sarcolemma. These granules appear to be at first of an albuminous, later of a fatty nature. The affected muscle appears yellow, soft, and friable. The heart-wall may retain its normal thickness, or it may be much atrophied. The greater part of the heart may undergo this change, or only small circumscribed portions, or only the superficial layers. The degeneration may lead to rupture of the heart, or to death from the inability of the heart to fulfil its functions. Phosphorus poisoning, typhus and severe fevers, pericarditis and exhausting diseases are found as causes for this condition, but it may occur in apparently healthy individuals.

Waxy or Amyloid Degeneration is found in connection with the same change in the other viscera, and is due to the same causes. The muscular fibres are the seat of the degeneration.

Mucous Degeneration of the subpericardial fat is found in persons much emaciated by chronic disease. The fat is changed into a translucent, gelatinous substance, composed of a homogeneous basement substance, coagulable by alcohol and acids, and of connective tissue-cells and compound granular corpuscles.

NEW GROWTHS.

Fibroma is sometimes seen in the form of small nodules growing in the heart-wall, or projecting under the endocardium or pericardium.

Lipoma is very rare in the form of a circumscribed tumor,

although a general increase of the subpericardial fat is common.

Myoma striocellulare.—Tumors formed of voluntary muscle have been seen in three cases as a congenital lesion. The tumors were single or multiple, some as large as a cherry, of the consistence and appearance of the surrounding heart-wall, but looking like circumscribed nodules. In one case, the tumor was cavernous. In one case, there were also gummy tumors in the heart-wall.

A single case has been observed, in an adult, in which the entire wall of the left ventricle was changed into a spongy mass, which may belong to the same category.

Syphilitic Gummata may occur at any point of the ventricular wall. They are usually multiple, may reach the size of a pigeon's egg, are of a whitish or yellow color, of fibrous or cheesy consistence; sometimes softer. They are composed of small round cells developed between the muscular fibres. The tumors may be almost entirely composed of cells, or may be partly cheesy, or mixed with fibrous tissue.

Tubercle are found in the heart-wall in the form of miliary granulations. They have also been observed in the valves.

Cysts containing serum, and others containing a grumous fluid, have been seen in rare cases.

Sarcoma sometimes occurs as a metastatic growth.

Carcinoma of the heart as a primary tumor is very rare. Nodules secondary to growths in the mediastinum or other regions are more common. Epithelial cancer in this organ is described by Paget and Virchow. Carcinomatous thrombi in the heart cavities sometimes occur as secondary formations from the same growth in other regions.

PARASITES.

Echinococci are sometimes found in the walls of the ventricles in their usual form of hydatid cysts.

Cysticercus sometimes occurs in connection with cysticerci in the voluntary muscles.

THE ARTERIES.

MALFORMATIONS.

The most important anomalies of the aorta and pulmonary artery have already been mentioned in connection with those of the heart. Congenital stenosis of the aorta will be spoken of under the head of stenosis. Anomalies in the distribution and origin of the arteries are principally of interest to anatomists.

DILATATION. ANEURISMS.

(1.) *Cirroid Aneurism* consists in the dilatation and lengthening of large or small arteries. The walls of the artery are thinned, the vessel is tortuous, and in places sacculated. These changes are most frequent in small arteries, especially the temporal and occipital; they involve the trunk of the vessel, and its branches, or may extend to the capillaries and small veins. They form larger or smaller tumors beneath the skin.

Rarely they are found in the larger arteries, and even in the aorta.

(2.) *The ordinary Aneurism* is a dilatation of the coats of the artery over a larger or smaller part of its course. Such dilatations are usually due to chronic endarteriitis. According to their shape we may distinguish two varieties: the diffuse and the circumscribed.

(1.) The diffuse, cylindrical, or fusiform aneurism consists in a uniform dilatation of all the coats of an artery, so that it assumes the shape of a fusiform or cylindrical swelling. In the walls of the dilated portion of the vessel there are often smaller circumscribed dilatations. The wall of the aneurism is atheromatous or calcified; the middle coat may be atrophied. The arch of the aorta is the most common seat of this form of aneurism; but the entire length of the aorta, or parts of any other arteries, may be dilated in the same way.

(2.) The circumscribed or sacculated aneurism consists either in a dilatation of the entire circumference of an artery over a short portion of its length, or in a dilatation of only a small portion of one side of the wall, so that the aneurism looks like

a swelling attached to one side of the artery. The aneurism commences as a dilatation of all the coats of the vessel; but as soon as it attains any size, the middle coat atrophies, so that the wall is composed of the inner and outer coats; or the inner coat is destroyed by endarteriitis, so that the outer coat alone forms the wall of the aneurism. As the aneurism increases in size, it presses upon and causes the destruction of the neighboring tissues and viscera, and portions of these tissues and viscera become incorporated with or take the place of the wall of the aneurism. The cavity of the aneurism is filled with fluid or clotted blood, or with layers of fibrine which adhere closely to its wall. The communication between the aneurism and the artery may be small or large. If arterial branches are given off from the aneurism, they may remain open, or become plugged with fibrine; or their walls are thickened and their cavities narrowed by endarteriitis. Death is produced by the pressure and interference of the aneurism with the adjoining viscera, or by rupture. The rupture may allow enough blood to escape to destroy life, or the blood may be held in by the soft parts, and a second false aneurism formed about the original one.

ANEURISMS OF THE DIFFERENT ARTERIES.

The Aorta may be dilated over its entire length; or there may be diffuse or circumscribed dilatations at any portion of its course; or there may be several aneurisms situated at different points.

The ascending portion of the arch of the aorta may be uniformly dilated in a fusiform shape; or there may be circumscribed dilatations on its anterior wall; or, more rarely, on its posterior wall. The sacculated aneurisms may be of all sizes, and may rupture within the pericardium; or they may form a cavity in the upper part of the ventricular septum, and communicate by openings into the pulmonary artery and left ventricle; or they may dilate downward between the visceral and parietal pericardium, in front of the heart, pushing that organ backward. They may perforate into the right or left auricle or right ventricle, the superior vena cava, or the pulmonary arte-

ry ; or they may reach a large size, press on and erode the right side of the sternum and adjoining ribs, project under the skin, and even rupture externally.

The transverse portion of the arch may be dilated in a fusiform shape ; or there may be sacculated aneurisms at any point in its wall. The sacculated aneurisms usually reach a considerable size. They press on the sternum and ribs in front, or on the œsophagus, trachea, and bronchi behind. The large arteries given off from the arch may be occluded. They cause death by pressure on the air-passages, the œsophagus, and the vena cava ; or may rupture externally, or into the œsophagus, trachea, bronchi, or pleuritic cavities.

On the thoracic aorta we find both diffused and circumscribed dilatations. They may reach a large size ; compress the ascending vena cava, the heart, œsophagus, bronchi, and lungs ; may erode the ribs, destroy the vertebræ, and open into the spinal canal. They may rupture externally, or into the bronchi, œsophagus, or pleural cavities.

On the abdominal aorta we usually find sacculated aneurisms. If they are situated high up, they may project into the pleural cavities ; if lower down, into the abdomen. They compress and displace the viscera, vessels, and nerves, and erode the vertebræ. They may rupture behind the peritoneum, in the peritoneal cavity, the pleural cavities, the inferior vena cava, the lungs, the colon, the pelves of the kidneys, the posterior mediastinum.

The Coronary Arteries may be dilated throughout, or may be the seat of small sacculated aneurisms. These may rupture into the pericardium, or may cause rupture of the heart-wall.

The Pulmonary Arteries are rarely the seat of aneurisms. Diffuse and circumscribed dilatations, however, sometimes occur on the main trunk and on the two principal branches of the artery. They usually do not reach a large size, but cause death by rupture. General dilatation of all the branches of the pulmonary artery is more common. It is found in connection with stenosis of the mitral valves, and with compression or induration of the lung tissue.

Of the other arteries of the body there is hardly any one which may not become the seat of an aneurism.

STENOSIS.

Stenosis and obliteration of the aorta, at the point of entrance of the ductus arteriosus, has been described in a considerable number of cases.

The situation of the stenosis is either exactly at the entrance of the ductus arteriosus, or close on either side of this point. The degree of stenosis varies. The aorta may be entirely closed and converted into a solid cord for a length of half an inch ; or there may be a circular constriction through which there is a larger or smaller opening. The constriction is uniformly circular ; or there is a septum springing from the concave side of the vessel at the opening of the ductus arteriosus ; or there is a cicatricial-like contraction of the aorta. The walls of the aorta at this point may be thickened and sclerosed. The ductus arteriosus may be closed or open. Above the constriction the aorta is usually dilated ; below it, it is normal, dilated, or stenosed.

Stenosis of the aorta produces hypertrophy of the left ventricle, and, later, of the right ventricle, with venous congestion throughout the body ; or there may be a collateral circulation developed between the arteries given off above and below the constriction ; or there may be rupture of the aorta, the right ventricle or auricle.

This condition is found at all ages, but is produced during foetal life, or in the first year of extra-uterine life. It is probable that it may be caused after birth by an abnormal closure of the ductus arteriosus. This vessel normally becomes closed without the formation of a thrombus. If a thrombus is formed, it may extend into the aorta and obstruct it ; or the ductus arteriosus is filled with a thrombus, but increases for a time in size ; afterward, as the thrombus is absorbed, the vessel contracts and draws the walls of the aorta together.

Stenosis of the aorta and of some of the other arteries has been observed in a few rare cases, without any known cause.

Endarteriitis, with the production of atheromatous and calcareous patches, may obstruct or entirely obliterate the smaller

arteries. This is especially seen in the arteries of the leg, foot, and brain, and in the coronary arteries.

Narrowing of the aorta, and of all its branches, with thinning of the arterial coats, is found as a congenital condition. It usually occurs in females in connection with want of development of the entire body.

Stenosis from thrombosis or embolism is treated of elsewhere.

RUPTURE AND WOUNDS.

Ruptures of arteries are found under the following conditions :

(1.) Fatty degeneration or endarteriitis, with atheromatous changes, may so soften and destroy the inner and middle coats of an artery as to admit of its rupture. The aorta, just above the valves, is the most frequent seat of this lesion. The rupture may run in any direction ; its edges are irregular and jagged. The blood may burst through all the coats of the aorta at the same point ; or, more frequently, the external coat remains, and the blood is infiltrated in the middle coat, and between it and the external coat. In this way, a dissecting aneurism is formed, which may extend along the aorta for a considerable distance. After a short time, the external coat gives way at some point, and the blood escapes. In rare cases, life is prolonged for some time, the rupture being closed by a new membrane.

We also find ruptures from fatty degeneration and atheroma in the arteries of the brain and of the lungs ; in the coronary arteries, the *cœliac* axis, and mesenteric arteries, and in the arteries of the upper and lower extremities.

(2.) In rare cases, stenosis of a portion of the aorta may cause rupture at some point between the seat of stenosis and the heart.

(3.) Contusions, wrenchings, and severe falls may rupture the wall of an artery either partially or completely, producing traumatic or dissecting aneurisms, or completely severing the vessel.

(4.) Penetrating wounds may wound or entirely sever an artery. If the artery be of large size, death from hæmorrhage is the usual result. A smaller artery may become closed, or be the seat of a false aneurism.

In the healing of a wounded artery, two conditions coöperate. The vessel retracts and contracts, and a thrombus is formed within it. The contraction may be alone sufficient to close the vessel; its coats thicken, and the inner surfaces finally are fused together; or the blood coagulates, and forms a thrombus in the vessel near the wound. This thrombus later becomes organized, and the vessel is converted into a fibrous cord.

Spurious aneurisms are found most frequently connected with vessels of the extremities. When an artery is wounded, the blood escapes into the surrounding soft parts, and a cavity is formed filled with blood and broken-down tissue. This condition may terminate in several ways.

(a.) The wound in the artery may heal, and the effused blood be absorbed.

(b.) The effused blood and broken tissues may become gangrenous and the surrounding soft parts be inflamed.

(c.) A sort of sac-wall may be formed by the soft parts, while the wound of the artery remains open, so that we have an aneurismal sac through which the blood is constantly pouring.

(5.) If an artery be wounded, and at the same time the vein which accompanies it, we have as the result the conditions called Aneurismal Varix and Varicose Aneurism. In aneurismal varix, the artery and vein become adherent at the seat of injury, so that the arterial blood passes directly into the vein. There is a smooth, rounded opening between the two vessels, the vein is dilated into a sac, and the veins emptying into it are dilated and tortuous.

In varicose aneurism, the artery and vein do not communicate directly, but a false aneurismal sac is formed between the vessels, into which the blood is poured before passing into the vein.

Varicose aneurism may also be produced by the spontaneous rupture of an aneurism into a vein. The aneurism presses against the vein, becomes adherent, and finally ruptures into it. This condition has been observed between the aorta and pulmonary artery; the aorta and inferior and superior vena cava; the popliteal artery and vein; the femoral artery and vein; the

splenic artery and vena azygos; the internal carotid and sinus cavernosus. Even in the cases of perforation by aortic aneurisms, life is usually prolonged for some time.

(6.) Destructive inflammation of the surrounding tissues may invade and destroy a portion of the wall of an artery. Thus ulceration of the trachea, bronchi, bronchial glands and œsophagus, may perforate the aorta; gangrene of the lungs, the pulmonary arteries; ulcer of the stomach, the gastric arteries, etc.

INFLAMMATION.

The walls of arteries are composed of three layers—the inner, middle, and outer coats. The inner coat, or tunica intima, consists of three layers: an inner layer of flat, thin, fusiform, or polygonal endothelium; a middle layer consisting of a finely granular basement substance traversed by fibres, and containing numerous fusiform and stellate cells; an outer layer composed in the smaller arteries of an elastic network; in the larger arteries, of one or more layers of an elastic, homogeneous fenestrated membrane.

The middle coat, or tunica media, is composed of involuntary muscular fibres, mostly running around the vessel, but some in its long axis; and of a variable amount of fibrous tissue, elastic tissue, and fenestrated membranes.

The outer coat, or tunica adventitia, consists of fibrous tissue and a network of elastic fibres.

The vasa vasorum or nutrient vessels of the arterial wall are distributed principally in the external coat, scantily in the middle coat.

Inflammation of an artery attacks principally the outer coat—*Periarteriitis*—or the inner coat—*Endarteriitis*.

Periarteriitis is not very common. It occurs in the larger arteries and is usually a secondary lesion. It is caused by injuries, by inflammation of the surrounding tissues, and by thrombosis or embolism. The outer and middle coats of the inflamed artery are congested and infiltrated, and thickened with a soft, grayish, structureless material. The cavity of the vessel is at first narrowed, afterward enlarged. At a later period, we find

various changes. The outer layers of the arterial wall may become necrotic, the inner coat may also die and be cast off in small fragments. In other cases, pus is produced either as a diffuse infiltration of the outer coats, or in the form of small circumscribed abscesses. The inner coat degenerates into little fragments, or is ruptured. In other cases, the inflammation has a more chronic character, and leads to fibrous thickening of the coats of the vessel, and frequently to its obliteration. In all the varieties, the presence of a thrombus in the artery, either as cause or effect of the inflammation, complicates the process. The thrombus may shrivel and be absorbed, or become organized, or be broken down and gangrenous, and form infarctions in other parts of the body.

Endarteritis is a chronic inflammation of the inner coat of arteries, resulting in the production of atheromatous and calcified plates. It rarely occurs before middle life, while in old age it is a very frequent senile change. The arch of the aorta is the most frequent seat of the lesion, but it may occur in any artery.

The disease begins in the form of small, flattened elevations on the inner surface of the artery. These elevations are formed by a hyperplasia of the outer layers of the inner coat. We find a considerable number of round, fusiform, and stellate cells with a basement substance, which is hard and fibrous or soft and mucous.

When the lesion has reached this stage, it goes on to fatty or calcific degeneration. Fatty degeneration attacks both the new cells and basement substance, converting them into a dense, yellow, cheesy mass; or into a soft, semi-fluid, yellow, puriform substance. Instead of cells and basement substance, we now find fatty and broken cells, granules, and plates of cholesterine. This condition is called atheroma. The softening process advances toward the inner surface of the tunica intima, covering the atheromatous patch, until a small perforation is formed and we find a small cavity filled with a thick, yellow fluid, communicating with the cavity of the vessel by a narrow sinus. As more of the intima is destroyed, the opening becomes larger, and we find a flat ulcer, often of considerable size. On the

roughened surface thus produced the fibrine of the blood is often deposited. The lesion may lead to the rupture of the vessel, or to the formation of ordinary, or of dissecting aneurisms.

Calcific degeneration takes place by the deposition of the salts of lime in the plate formed by the hyperplasia of the intima. We find thin plates, looking like bone, still covered by the intima, or bare. The plates may be few and small, or large and numerous. The process may be accompanied by a calcification of the muscular cells of the middle coat. All the stages of simple hyperplasia, fatty, and calcific degeneration are often met with in the same vessel.

Simple fatty degeneration of the intima may occur in the same artery in which the lesions of chronic endarteriitis are found.

Under the name of Canalization Rokitansky describes a rare change in the inner coat. The intima is thickened and pierced with numerous small canals, into which the blood passes. In this way, the arterial wall is converted into a spongy, cavernous tissue resembling the corpus cavernosum.

DEGENERATION.

Simple Fatty Degeneration affects only small, circumscribed portions of the inner coat. There is a fatty degeneration of the cells of the intima, with absorption of the basement substance. The process begins in the inner layers of the intima, and gradually works outward, forming small excavations. It may lead to rupture of the vessel.

Calcification of the muscular cells of the middle coat occurs in connection with atheroma, with caries of the bones, and as an idiopathic affection. It is usually found in old people, and involves arteries of medium size. It may lead to gangrene of the parts supplied by the affected vessels.

Amyloid Degeneration of the arteries is of common occurrence. The inner coat and the muscular layer are infiltrated with a translucent material looking like wax. In extreme cases, the outer coat is infiltrated in the same way. The walls of the vessel appear thickened, stiff, and of a peculiar waxy color, they are colored mahogany red by the addition of a weak solution of Iodine. The lumen of the affected vessel is diminished in size.

THE VEINS.

DILATATION.

Dilatation of the Veins, or Phlebectasia, presents itself under a variety of forms.

(1.) Simple Dilatation. The vein is uniformly dilated in a cylindrical or fusiform shape; its length is not increased; its walls are of normal thickness, or thinned; the valves increase in size, or are insufficient, or atrophy, or are torn.

(2.) Cirroid Dilatation. The vein is uniformly cylindrically dilated, but is also increased in length, so that it assumes a very tortuous course. The walls are normal, thickened or thinned.

(3.) Varicose Dilatation. A circumscribed portion of the wall of the vein is dilated so as to form a globular sac. The sac communicates with the vein through a large or small opening. The wall of the sac is formed of the coats of the vein, which preserve their normal thickness, are thickened or thinned; the middle coat may disappear entirely. There may be only one such dilatation, or there may be a number on the same vein, or a number of veins may be affected at the same time. The vein may be otherwise normal, or, more frequently, is dilated in the cirroid form.

(4.) Anastomosing Dilatation. A number of contiguous and anastomosing veins are dilated, both in the cirroid and varicose forms. The vein then looks like a series of cavities separated by thin partitions. The dilatations of the same vein become adherent to each other and to those of the adjoining veins; portions of the wall of the dilated parts may disappear, and we find a number of cavities containing venous blood, and separated from each other by thin partitions. The course of the vein can no longer be followed out.

Spontaneous cure of dilatations of the veins is not common, and usually occurs only in the lesser degrees of the lesion. Most phlebectasiæ increase steadily in size and extent. Very frequently thrombi form in the dilated veins, and either partially or completely fill them. The wall of the dilated sac may

become so thin that it finally ruptures, and the blood is discharged externally. Sometimes inflammation is set up in the tissues surrounding the vein, and we find both the surrounding tissues and the wall of the vein the seat of purulent infiltration or fibrous thickening. The parts of the body from which the dilated veins draw their blood exhibit the results of chronic venous congestion, œdema, hyperæmia, and hypertrophy.

There is hardly one of all the veins of the body which may not be dilated. The veins of the leg and thigh; those of the pelvis and pelvic viscera; those of the spermatic cord, scrotum, and labia; those of the abdominal wall; those of the neck and arm are the ones most frequently found in this condition.

The causes of dilatation are some mechanical obstruction to the passage of the blood through the veins toward the heart.

WOUNDS. RUPTURE.

Wounds of the veins usually heal by a simple contraction, and an adhesive inflammation of their walls; sometimes by the formation of a thrombus. Rupture of the veins is produced by severe contusions and crushings of the body, and by violent falls.

Perforation of a vein is produced by suppuration of the soft parts and invasion of the walls of the vein; by suppurative inflammation beginning in the wall of the vein; by the pressure of an aneurism or of a new growth; by thinning of the venous wall in phlebectasie.

INFLAMMATION.

Inflammation of a vein may affect principally the outer coat—periphlebitis—or the inner coat—endophlebitis.

Periphlebitis is of not infrequent occurrence. It is caused by the presence of a thrombus, by injuries, and by inflammation of the surrounding tissues. It may be of a suppurative or of an indurative character. In suppurative periphlebitis, the outer layers of the venous wall are congested, swollen, infiltrated with serum and pus-cells. The inner coat degenerates, and portions become necrotic, and may be cast off. In this way, the vein may be perforated either from the inside or the out-

side. A thrombus in the vein is the constant accompaniment of periphlebitis. It usually is the cause of the inflammation, but may be its result. The changes which take place in the thrombus, and the infarctions produced by its fragments in different parts of the body, are the most important results of phlebitis.

Periphlebitis may terminate in absorption of the thrombus, with the return of the vein to its normal condition; in obliteration of the vein; or in the death of the patient from infarctions.

Chronic Periphlebitis produces thickening of all the coats of the vein, but principally of the outer coat. The surrounding soft parts are also thickened, so that the vein can hardly be separated from them. The lumen of the vein remains open, or is filled with a thrombus.

Endophlebitis is an inflammation of the inner coat of the vein, of the same character as chronic endarteriitis. Circumscribed patches of thickening of the inner coat are produced, which afterward undergo fatty and calcareous degeneration.

Waldeyer has lately drawn attention to the fact that the division of the walls of the veins into three coats, as in the arteries, is an artificial one. The venous wall is rather a layer of connective tissue, covered on its inner surface with endothelium, and reinforced in places by muscular fibres and elastic tissue.

If the outer part of the venous wall is inflamed, the morbid process is apt to extend to its inner surface. We then find on the internal surface of the vein very fine velvet-like granulations, which are formed by an increase of the endothelial cells. These granulations may increase in size until the wall of the vein is covered with a soft mass, which can hardly be distinguished from a thrombus. The mass is, however, a soft, organized, vascular granulation tissue, continuous with a similar cell growth through the entire thickness of the wall of the vein.

The form of phlebitis is attended and followed by the formation of a thrombus.

NEW GROWTHS.

Myoma has been observed in two cases: one on the saphenous vein, and one on the ulnar vein. The tumors were of small size, and formed by a circumscribed hypertrophy of the muscu-

lar coat of the vein. The corresponding portion of the lumen of the vein was dilated. The growth appeared like a globular dilatation of the vein, with great hypertrophy of the muscular coat.

Myo Sarcoma has been observed in one case. The tumor was as large as a man's fist, situated in the cavity of the dilated vena cava, extending from the liver to below the renal veins. The growth had invaded the adjoining portion of liver, and there were secondary growths in the lymphatic glands. The tumor consisted of connective tissue, smooth muscle, fusiform cells, and myeloplaxes.

Carcinoma of the surrounding parts may perforate the wall of a vein, and grow in its cavity, so as to form a thrombotic mass of considerable size. Cases have been reported in which the new growth was confined to the cavity of the liver veins, and not found in any other part of the body.

PARASITES.

Echinococcus is sometimes found in the veins, either developed there, or having perforated from the adjoining tissues.

Distoma hepaticum is found in the veins of the liver.

Distoma hæmatobium has been found in the portal vein and its branches, principally in Egyptians.

THE ALIMENTARY CANAL.

THE MOUTH.

MALFORMATIONS.

Malformations of the lips and cheeks are usually associated with defective formation of the bones of the mouth. The entire process is generally due to an arrest of development.

(1.) The lower jaw is absent; the upper jaw and hard palate small and imperfectly formed; the temporal bones nearly touch in the median line. The lower half of the face is therefore wanting; the mouth is absent, or small and closed posteriorly;

the tongue is absent. Such a malformation is rare; the fœtus is not viable.

(2.) The face remains in its early fœtal condition of a large cleft; the mouth and nose form one cavity; the orbits may be united in the same cavity. The fœtus is not viable.

(3.) There is a cleft in the upper lip, upper jaw, and hard palate. The cleft corresponds to the point of junction of the processes of the superior maxilla with the intermaxillary bone. There may be one cleft or two, one on either side of the intermaxillary bone. The cleft involves the lip alone; or the lip and superior maxilla; or the lip, maxilla, and palate. There may be a single or a double cleft in the palate; and the cleft may involve either the hard or soft palate, or both. If there are two clefts of the lip and maxilla, the portion of lip and bone between them may be small, or entirely absent, so as to leave a large open space. The soft palate may be entirely absent. This is a common malformation, and does not endanger life.

(4.) Rarely we find a cleft involving the middle of the lower lip, and sometimes extending into the inferior maxilla.

(5.) Either the inferior, the superior, or both maxillary bones may be abnormally small.

(6.) The edges of the lips may be partly or completely joined together. The opening of the mouth may be only a round hole.

(7.) The lips may be absent, or imperfectly developed.

(8.) The corners of the mouth may be prolonged by clefts in the cheeks nearly to the ears.

HYPERTROPHY.

The skin of the cheeks and lips may be hypertrophied in connection with elephantiasis of the face.

There may be a thickening of the lips alone, so that they appear double. This thickening may be due to an increase of all the anatomical elements of the lips; or there may be an increase and dilatation of the lymphatic vessels, giving to the growth a soft, œdematous character.

INFLAMMATION.

1. *Catarrhal Stomatitis* is seldom an object of post-mortem importance, except when caused by irritating poisons. It is found most frequently in children, but also in adults. It is produced by a great variety of local and constitutional causes.

Of the conditions which are seen during life, the hyperæmia, increased secretion, white coating and swelling of the mucous membrane, but little remains after death.

If irritating poisons have passed through the mouth, the mucous membrane, especially near the pharynx, will be found intensely red, or with black or white patches surrounded by intense congestion.

2. *Croupous Stomatitis* is caused by local irritants; by a continuation of the same disease from the larynx and pharynx; as a result of syphilis and mercurial treatment; with the exanthematous fevers; in cachectic subjects in the form of aphthæ; as epidemic diphtheria. The mildest degree of this form is seen in the aphthous sore mouth of children and cachectic persons. Numerous little white patches of fibrinous exudation make their appearance on the lips and gums. The exudation is cast off with the epithelium which adheres to it, leaving a superficial ulcer, and this ulcer may be again covered with fresh exudation.

The severest degrees are found in endemic diphtheria. Fibrinous exudation is found both on the surface and in the substance of the mucous membrane. In consequence of this, large patches of the mucous membrane become gangrenous, and are cast off. The gangrene may extend also to the subjacent soft parts, and form deep ulcers. If the patient recovers, these ulcers cicatrize, and may, by their contraction, produce great deformities.

3. *Phlegomonous Stomatitis* is caused by injuries, by diseases of the teeth and bones, and may be idiopathic. The inflammation attacks the deep layers of the mucous membrane, and forms abscesses there. Sometimes there is so much swelling of the soft parts about the fauces, that there is imminent danger of suffocation.

ULCERATION.

Simple follicular ulcers of the mucous membrane of the mouth are common and trivial. Syphilitic ulcers may be merely superficial, or they may be produced by the softening of gummy tumors in the deep layers of the mucous membrane, and attain a large size. Sometimes chancres are found in this situation.

Scorbutic ulcers may be preceded by croupous inflammation, or by simple gangrene of small portions of the mucous membrane. They may be extensive and of a serious character.

GANGRENE.

Gangrene of the lips and cheeks, or Noma, is most frequent in cachectic children, and as a consequence of the abuse of mercury. Much more rarely it occurs in adults after typhus and other exhausting diseases. The disease begins in mucous membrane of the cheeks, near one of the corners of the mouth. The mucous membrane becomes infiltrated with pus, discolored, black, and gangrenous. The same rapid gangrenous process extends through the entire thickness of the cheek, and produces perforation. At the same time the gangrene spreads in all directions, involves the neighboring soft parts, the bones, and even the neck.

Besides this form of gangrene, other forms are produced by erysipelas, burns, bites of insects, and glanders.

NEW GROWTHS.

Adenoma occurs in the mucous membrane covering the mouth, lips, and soft palate. The tumors are rounded, usually small, but sometimes as large as a hen's egg. They may be situated in the deep layers of the mucous membrane, or project in a polypoid form. They are formed by an hypertrophy of the normal mucous glands. The glandular acini are increased in number and size; the epithelial cells are increased in number, and often undergo colloid degeneration, so as to form cysts.

Papilloma occurs most frequently at the edges of the lips,

but is also found on the gums, the floor of the mouth, and the cheeks. The growths look at first like warts, and are formed of hypertrophied papillæ covered by thickened epidermis. They have a marked tendency to ulcerate, and may then become the starting-point of epithelial cancer.

In some cases, the growth of epidermis becomes the principal feature in the process. Over one or more hypertrophied papillæ, an immense growth of epidermis takes place, so as to form projections over an inch in length, the so-called cornu cutaneum.

Epithelioma occurs most frequently on the edge of the lower lip, but has been observed at many other parts of the mouth. It begins either as an ulcerating papilloma, or as a small nodule deep in the mucous membrane. In either case, a portion of the mucous membrane becomes replaced by an indurated, ulcerating tissue. The disease may spread over a large part of the lips, the cheeks, the floor of the mouth, tongue, and pharynx. The neighboring glands are involved, and metastatic tumors may be formed in other viscera.

Carcinoma is said to occur, in rare cases, in the lips and palate, and to assume there the medullary form.

Angioma is frequent on the lips, rare on the palate; it may be congenital or developed after birth.

Fibroma, *Lipoma*, *Enchondroma*, have been seen in a few cases in the lips. When they appear in the mouth, they usually grow from the bones.

THE TONGUE.

MALFORMATIONS.

Absence of the tongue is found in connection with the extreme defects of development in the face already mentioned.

The anterior portion of the tongue may be absent while its base remains. The lower jaw is then small.

The tongue may be partly or completely adherent to the floor of the mouth. The frenulum may be abnormally short, or may extend to the tip of the tongue. In rare cases, the sides of the tongue are adherent; or its upper surface may be adherent to the roof of the mouth.

Very rarely the tongue is separated into two halves by a cleft, extending from the tip to the root.

There may be two tongues, one situated over the other.

HYPERTROPHY.

Makroglossia, or hypertrophy of the tongue, is usually a congenital, sometimes an acquired condition. The congenital form continues to increase after birth. The jaws, lips, and cheeks are deformed by the pressure of the enlarged tongue.

The tongue may reach an enormous size. Its surface remains smooth, or is nodulated and fissured. The increase in size appears to be due principally to a growth of connective tissue. The muscular fibres retain their normal development. The lymphatic vessels are often enlarged. The epithelium is hypertrophied. The hypertrophy is not always uniform; part of the tongue may be larger than the rest.

INFLAMMATION.

Inflammation may involve the mucous membrane, or the interstitial tissue of the tongue. Inflammation of the mucous membrane forms a part of the different forms of stomatitis.

Parenchymatous Glossitis is produced by injuries of the tongue, by mercury, typhus fever, small-pox, etc. It usually runs an acute course, and involves the entire tongue. The tongue is swollen, red, fissured, covered with crusts or fibrinous exudation.

The swelling may be so great as to cause suffocation and death; or it may go on to suppuration or to gangrene; or become chronic, and result in fibrous induration.

NEW GROWTHS.

Cysts.—The most common forms of cyst are the large sacs beneath, or partly in the substance of the tongue, (Ranula.) Some of these are dilatations of the ducts of the submaxillary and sublingual glands; others are new growths in the connective tissue.

Rarely cysts are found entirely in the tongue itself.

Small cysts are sometimes found in the mucous membrane of the tongue.

In the floor of the mouth, beneath the tongue, dermoid cysts have been observed.

Angioma.—Cavernous vascular tumors are found in the substance of the tongue, and projecting from its surface.

Lipoma and Fibroma are rare. They may occur as nodules, or project in a polypoid form. Composite tumors, largely composed of fat, are found in the tongue as a congenital condition.

Tubercles are found in rare cases in the form of ulcers, of which the walls are infiltrated with miliary tubercles.

Syphilitic Gummata occur as white nodules in the intermuscular fibrous tissue.

Lupus occurs in the form of nodules and of ulcers at the base of the tongue.

Sarcoma in this situation appears not to have been described. I have seen one such tumor in a young child. It formed a nodule as large as a chestnut, and was composed of round and fusiform cells.

Epithelioma may begin as a primary growth in the tongue; or the growth may invade it from the adjoining tissues. It may destroy a large part of the organ, invade the floor of the mouth, and form secondary tumors in the adjoining lymphatic glands.

THE PHARYNX AND THE ŒSOPHAGUS.

MALFORMATIONS.

Fistulous openings from the pharynx, running between the muscles, and perforating the skin, have been seen in rare instances. These fistulæ are so small as to have no practical interest.

The œsophagus may be entirely absent, or its lower portion may be present, and joined to the pharynx by a solid cord; or the pharynx, or the lower part of the œsophagus, may be continuous with the trachea; or the entire œsophagus may be represented by a solid cord.

Diverticula of the pharynx, dilatations of the œsophagus, and

division of the middle portion of the œsophagus into two branches have all been observed.

INFLAMMATION.

Catarrhal and Croupous Pharyngitis are usually associated with the same forms of inflammation in the mouth, and have the same characters.

Submucous Pharyngitis may occur with inflammations of the mucous membrane, with caries of the cervical vertebræ, with inflammation of the cervical and parotid glands, with periostitis of the cranial bones, or may be idiopathic. It may result in swelling and œdema, in induration, or in suppuration. It is most important when it affects the posterior wall of the pharynx, and forms retro-pharyngeal abscesses. Such abscesses may cause death by suffocation.

Catarrhal Œsophagitis may be either acute or chronic. The chronic form may produce ulceration, or relaxation and dilatation of the walls, or hypertrophy of the muscular coat.

Croupous Œsophagitis is found with croup of the pharynx, and after the exanthemata and other severe diseases.

Irritating and caustic acids and alkalies destroy larger or smaller portions of the mucous membrane. The necrosed portions are of a black or whitish color, surrounded by a zone of intense congestion. If the patient recover, the patches of membrane which have been destroyed slough, fall off, and leave a granulating surface. In this way, dangerous stenosis of the œsophagus may be produced.

Foreign bodies, which are swallowed and become fixed in the œsophagus, cause inflammation of the mucous membrane and of the adjoining soft parts. The inflammation may go on to produce abscesses around the œsophagus, or to destroy the wall of the canal, and the foreign body finds its way into the trachea, aorta, or pericardium.

Inflammation of the submucous tissue of the œsophagus, apart from the cases just mentioned, is not common. It may cause the formation of abscesses, or of fibrous masses, which may produce stenosis.

ULCERATION.

Ulceration of the Pharynx occurs in rare cases as the result of catarrhal inflammation. More frequently it is produced by syphilis, either in the form of superficial ulcers, or of deep and extensive destructions of tissue from the softening of gummy tumors. Lupus, also, sometimes attacks the upper part of the pharynx.

Ulceration of the Œsophagus is not common, and but little is known concerning it. Some authors describe a simple perforating ulcer, similar to that of the stomach. Foreign bodies in the œsophagus may perforate its wall, as already mentioned. Perforations of the œsophagus from without may be produced by inflamed bronchial glands, by cavities and gangrene of the lungs, by emphysema, by abscesses in the mediastinum, by abscesses accompanying caries of the vertebrae, and by aneurisms of the aorta.

Cases have been described of rupture of the normal wall of the œsophagus by violent coughing and vomiting; but it seems probable that there was really some previous disease.

DILATATION AND STENOSIS.

The œsophagus may be dilated throughout its entire length, or at some one point. The general dilatation is most frequently caused by stenosis of the cardiac orifice of the stomach. The wall of the œsophagus is usually hypertrophied.

Less frequently the entire œsophagus is dilated, without any satisfactory cause to account for it. The dilatation may be so great that a man's arm can be introduced into the cavity of the œsophagus. Its walls are then usually thickened, but sometimes thinned.

The circumscribed dilatations may be fusiform or sacculated.

The fusiform dilatations are congenital, or are produced by a stenosis at any part of the canal.

The sacculated dilatations are situated at the upper part of the œsophagus or the lower part of the pharynx. They begin as a small pouch in the posterior wall and at right angles to it. Such

a pouch becomes longer as the food accumulates in it and hangs down behind the œsophagus. The pouch then compresses the œsophagus, and its mouth forms nearly a straight line with the œsophagus above it, so that the food passes more readily into the sac than into the œsophagus below it. In this way, the sac is constantly distended with food, from which it is only relieved by occasional vomiting. In this way, the dilatation goes on increasing until the patient dies from starvation.

The causes of this condition are: portions of solid food remaining for some time in the œsophagus or in a fold of its mucous membrane; the traction of adhesions between inflamed lymphatic glands and the œsophagus; stenosis and hernial protrusions of the mucous membrane of the pharynx through spaces in its muscular coat.

Stenosis of the œsophagus is produced by changes in its walls or by external pressure.

Changes in its walls are produced by new growths, by cicatrices, by thickening of the submucous connective tissue, and by hypertrophy of the muscular coat.

Pressure from without is produced by tumors in the neck and thorax; by enlarged glands; by aneurisms; by exostoses of the vertebræ; by the right subclavian artery, when it arises from the arch of the aorta on the left side and passes between the œsophagus and the vertebræ, or between the œsophagus and trachea; by abscesses in the neck and mediastinum; by tumors and abscesses in the lungs, liver, and diaphragm.

NEW GROWTHS.

Epithelioma is the form of malignant disease which most frequently attacks the pharynx and œsophagus, although true cancer is said to occur there.

The upper part of the pharynx is seldom the primary seat of the disease. Usually it is invaded by the disease beginning in the mouth, the cervical glands, or the base of the cranium.

In the lower part of the pharynx and the œsophagus the disease is usually primary. The new growth may extend along the wall of the œsophagus for a considerable distance, but more frequently encircles it for a space of from one to five inches.

The growth begins in the submucous tissue. We then find a hard, prominent ring encircling the œsophagus and producing a greater or less degree of stenosis; after a time, the mucous membrane and the new growth ulcerate, and instead of the stenosis there is a cavity with ragged walls. Or, the mucous membrane ulcerates at an early stage of the disease, and the growth of the epithelioma does not exceed its destruction by ulceration. Then we find an ulcer encircling the œsophagus, of which the malignant character can be easily overlooked. More rarely the mucous membrane ulcerates and the pseudoplasm grows rapidly and projects in fungous masses into the cavity of the œsophagus.

If the new growth involves the entire thickness of the œsophageal wall, it may go on to invade the neighboring parts and may perforate the trachea, the bronchi, the lungs, the aorta, the pulmonary artery, or the vertebræ. Secondary and metastatic tumors are very rare.

Fibroma occurs in the pharynx in the form of small polypoid tumors. These little polypi are covered with mucous membrane, while their interior is made up of a loose, vascular fibro-cellular tissue.

Besides these, large polypoid tumors make their appearance in the pharynx and posterior nares, which grow from the periosteum of the bones of the base of the skull and of the upper cervical vertebræ. These tumors push the mucous membrane before them, often reach a large size, and may prove fatal.

Sarcomata also grow from the periosteum of the neighboring bones and project into the pharynx.

Lipoma is sometimes found in the wall of the œsophagus, forming small nodules in the submucous tissue.

Cysts are sometimes formed in the walls of the pharynx and œsophagus by dilatation of the mucous glands.

Myoma occurs in the form of hard, rounded tumors growing from the muscular coat. The tumors usually project inward, and sometimes even have a polypoid shape.

THE STOMACH.

MALFORMATIONS.

Malformations of the stomach are not common. The organ may be entirely wanting in acephalous foetuses. It may be of various degrees of smallness, sometimes no larger than the duodenum. It may be divided into two halves by a deep constriction in the middle. The pyloric orifice may be stenosed or entirely closed. The stomach may be outside of the abdominal cavity from a hernial protrusion through the diaphragm, or at some point in the abdominal wall. It is found on the right side instead of the left, when the other viscera are transposed, and the position of the cardiac and pyloric orifices is correspondingly inverted.

POST-MORTEM CHANGES.

In adults, the stomach after death presents a grayish or pinkish color, mottled with red ecchymoses. The mucous membrane is soft, and the epithelium easily brushed off. At the fundus the food is usually found collected, and here the mucous membrane is the softest. It is very common to find the epithelium removed from the entire fundus of the stomach, so that all that portion of its wall is grayer and thinner, there being a sharp dividing-line between the two portions. Sometimes this post-mortem softening process goes on to destroy all the coats of the stomach, and even the adjoining portion of the diaphragm. In this way, the contents of the stomach may be emptied into the pleural cavity by a large, ragged opening in the stomach and diaphragm. When the softening affects all the coats of the stomach, the softened portion is not sharply limited. The entire thickness of the affected portion of the wall is converted into a gray or yellow semi-transparent jelly, or into a blackish, broken-down pulp.

This softening is most frequent in children, but also occurs in adults, usually in connection with severe and exhausting diseases.

There have been and are many different opinions as to the nature of this softening process. Some believe it to be an

ante-mortem disease with regular symptoms. Some distinguish an ante-mortem and a cadaveric softening, but acknowledge that it is not easy to distinguish between the two after death. Others hold that the process is entirely a post-mortem one, but are divided as to whether the softening is produced by the gastric juice or is a mere putrefactive change induced by the presence of food in the stomach. All these points can hardly even now be said to be settled. The weight of evidence, however, seems to be in favor of the post-mortem and putrefactive nature of the softening.

INJURIES.

Perforating wounds of the stomach cause, as a rule, fatal peritonitis. In rare cases, however, the patient recovers with a gastric fistula, or even with complete closure of the wound.

Rupture of the stomach is sometimes caused by severe blows and falls. All the coats of the organ are torn through, or they are separated from each other by extravasated blood.

Cases of spontaneous rupture of the stomach from over-distension with food have been reported. Most, if not all, of them were probably really perforating ulcers.

HÆMORRHAGE.

An effusion of blood may take place in the cavity, or in the walls of the stomach. The blood may come from the mouth, œsophagus, or trachea, and be swallowed. Or it comes from the vessels in the wall of the stomach.

The most frequent causes are: ulcers; cancer; obstruction to the current of venous blood from diseases of the liver, heart, and lungs; poisons and foreign bodies; dyscrasiæ, as scurvy, yellow and relapsing fever; inflammation of the stomach; and vicarious hæmorrhages.

When the hæmorrhages are in the cavity of the stomach, the organ is found to contain blood, either in clots or mixed with food, and converted into a black, grumous fluid. Not infrequently, however, the blood is vomited during life, or passes into the intestines, and the stomach is left empty.

When the blood escapes from large vessels destroyed by

ulcers or cancer, the source of the hæmorrhage is easily discovered. But when the bleeding is from the capillaries in consequence of obstruction to the portal circulation, no lesions are to be found in the mucous membrane, excepting sometimes ecchymoses.

In relapsing fever, scurvy, purpura, and such diseases, the hæmorrhage may be in the wall of the stomach, which then presents a mottled red and gray color, or the superficial layers of the mucous membrane soften and fall off over such hæmorrhages, leaving little depressions—hæmorrhagic erosions.

INFLAMMATION.

Acute Catarrhal Gastritis is but seldom seen in autopsies, and does not present constant lesions. The mucous membrane may be red and swollen, covered with a layer of mucus and epithelium; or the hyperæmia, which existed during life, may disappear after death, and the stomach present no lesions.

Chronic Catarrhal Gastritis is, on the other hand, very common, but its lesions are not always pronounced. In the advanced cases, the mucous membrane is red, white, or slate-colored, and thickened. The thickening is uniform, or it is punctate, giving the appearance of numerous little prominences—*état mamelonné*. The gastric follicles atrophy, and the connective tissue between them is increased. At the same time the muscular coat is hypertrophied, and the thickening may be so great as to cause stenosis of the pylorus. Sometimes there are polypoid outgrowths from the mucous membrane, and sometimes its glands become cystic. Less frequently hæmorrhagic erosions and ulcers are also found.

In the lesser degrees of the disease, the mucous membrane is congested uniformly or in spots; or it is white and anæmic, or of an irregular slate-color. The pyloric portion of the stomach is the most frequent seat of these different lesions.

Chronic catarrhal gastritis is either a sequence of acute catarrhal inflammation and due to the same causes—changes of temperature, irritating food or drugs, alcoholic drinks, and chronic diseases, such as phthisis—or it is caused by obstructions to the venous circulation in the liver, heart, or lungs.

The pressure of an aneurism of the arch of the aorta on the ascending vena cava may cause an intense and uniform congestion of the mucous membrane, which can hardly be distinguished from acute gastritis.

Croupous Gastritis is of rare occurrence. It is found in children with croupous inflammation of the pharynx and œsophagus, and is then usually in small patches. In adults, it is almost always secondary to typhus, pyæmia, puerperal fever, cholera, dysentery, the exanthemata, and irritating poisons. The false membrane is in small patches, or may line a large part of the stomach. The disease is usually not diagnosticated during life, the symptoms of the primary disease diverting attention from the gastritis.

I have seen one case of idiopathic croupous gastritis in an adult. A man, 46 years old, was in good health until eight days before his death. At that time he caught cold, had pains over his bowels, tenderness over the liver, constipation, cough with mucous expectoration, temperature $102\frac{1}{2}^{\circ}$, pulse 120. On the day of his death, the 8th day of the disease, the temperature was 100° , pulse 112, tongue dry, abdomen tympanitic and tender, and he died in a prolonged attack of syncope. At the autopsy all the viscera were examined. Excepting evidences of bronchitis in the lungs, there were no lesions save in the stomach. About $\frac{2}{3}$ of the internal surface of the stomach, including the lesser curvature and anterior and posterior walls, appeared to be covered with a thick false membrane, which did not quite reach to the cardiac or pyloric orifices. Minute examination showed that there was a layer of exudation on the internal surface of the mucous membrane. This exudation consisted of fibrillated fibrine and lymphoid cells, dipping into the mouths of the follicles. Beneath the exudation the mucous membrane was thickened and altered. A large number of lymphoid cells separated the follicles, and even replaced them entirely. The submucous layer was very much thickened by the presence of lymphoid cells, fibrillated fibrine, and fibrous tissue. The muscular coat was separated into layers by groups of lymphoid cells.

Parenchymatous Gastritis, Gastradenitis, Parenchymatous

degeneration of the gastric tubules. This condition is found with many severe diseases—Scarlatina, Measles, Typhus, Phosphorus poisoning, Small-Pox, etc. The mucous membrane of the stomach is congested, uniformly or in patches, and swollen. The cells in the gastric tubules are enlarged, granular, and distend the tubules. In the more chronic cases, there is no congestion. The mucous membrane is opaque, thick, and of a yellowish color. The tubules are distended with granular cells.

Phlegmonous Gastritis is very rare. It may be secondary or idiopathic. In the latter case, it may cause death in a few days, with the symptoms of acute peritonitis. The wall of the stomach is found thickened by exudation in its submucous coat. This exudation may run out, on section, as a yellow fluid of various degrees of density; or it remains in place as a soft, sticky, yellow material. These different appearances depend upon the relative proportion of lymphoid cells, fibrine and serum. The exudation may extend over part, or the entire extent of the wall of the stomach. It may break through the mucous membrane by small openings. There may be at the same time general peritonitis.

Toxic Gastritis.—The mineral acids, the caustic alkalies, arsenic, corrosive sublimate, and the metallic salts, phosphorus, camphor, and all other irritating materials, cause different lesions of the stomach, according to their quantity, their strength, and the length of time that has elapsed before death.

In large quantities, they destroy and convert into a soft, blackened mass both the mucous membrane and the other coats, so that perforation may take place. In smaller quantities, they produce black or white sloughs of the mucous membrane, surrounded by a zone of intense congestion. If death does not soon ensue, the ulcerative and cicatricial processes which follow such sloughs, may contract and deform the stomach in various ways.

If the poisons are of less strength, they produce a diffused congestion of the mucous membrane, with catarrhal or croupous exudation on its surface, and serous infiltration of the submucous coat.

ULCERATION.

The Simple Perforating Ulcer of the stomach is a lesion frequently seen. It occurs in young persons, especially females. It may cause death by starvation, hæmorrhage, or peritonitis; or may cicatrize completely, and the patient recover.

These ulcers are usually single, but there may be several of them. They occur most frequently in the pyloric portion of the stomach, at some point in the posterior wall near the lesser curvature, but they may be found at any portion of the stomach.

The ulcers are usually circular, sometimes oval, annular, or of irregular shape; or two ulcers may combine to form an irregular figure. The perforation is largest in the mucous membrane, smallest in the peritoneum. The edges of the ulcer present no changes, or, if the ulcer is of long standing, are thickened and indurated. The rest of the stomach is unchanged, or is in a condition of chronic catarrhal inflammation.

The ulcer may perforate directly through the walls of the stomach; the contents of the organ are discharged into the peritoneal cavity, and fatal peritonitis supervenes. This is especially the case when the ulcer is in the anterior wall. Or adhesions are formed between the stomach and the neighboring viscera, so that the bottom of the ulcer is sealed. Or, if the liver, the intestines, or the abdominal wall become adherent, they are invaded; an ulcerating cavity is formed in the liver, a fistulous communication is established between the stomach and the intestines, or the outer surface of the abdomen. Or the adhesions are only partial, and an abscess is formed about the perforation. Such an abscess may remain shut in by adhesions, or may break into the cavity of the peritoneum, and cause peritonitis. During the progress of the ulcer, there may be repeated small hæmorrhages from the erosion of small blood-vessels, or large and fatal hæmorrhages if large trunks are destroyed.

In many cases, these ulcers completely cicatrize, leaving a stellate scar; or, if they are large, constricting the stomach in various ways.

The way in which these ulcers are formed is very obscure, and the explanations usually given are theoretical rather than based on actual observation. It is said that they are preceded by a circumscribed stasis and hæmorrhagic infiltration of a portion of the stomach-wall, and that the part thus infiltrated afterward sloughs, and leaves the ulcer. Such a stasis may be caused : (1) by catarrhal gastritis ; (2) by embolism, or thrombosis of an artery of the stomach ; (3) by chronic or acute venous hyperæmia, especially from thrombosis of the gastric and portal veins ; (4) by fatty and atheromatous degeneration of the blood-vessels.

Hæmorrhagic Erosions occur as rounded spots or narrow streaks, formed by a loss of substance of the mucous membrane. The mucous membrane at these points is congested, soft, and covered by small blood-clots. The destruction of the mucous membrane is usually superficial, but may involve its entire thickness. The number of these erosions may be so great that the entire internal surface of the stomach is studded with them. They give rise to repeated hæmorrhages, and are accompanied by catarrhal inflammation of the rest of the mucous membrane.

They occur at all periods of life, even in infants. Their usual seat is the pyloric portion of the stomach.

They may be idiopathic. Usually, however, they occur in connection with some serious general disease.

Catarrhal Ulcers are formed with chronic gastritis. They are small, round, or irregular, superficial or deep, congested, and usually in small numbers. They may cicatrize or become perforating ulcers.

Follicular Ulcers, formed by inflammation and ulceration of the solitary follicles are very rare. They occur most frequently in young infants.

DILATATION AND STENOSIS.

Dilatation of the stomach is spontaneous or produced by stenosis.

When produced by stenosis at the pylorus, the fundus is first dilated, afterward the entire stomach. If the stenosis is at another part of the stomach, that portion of the organ which is

between the stricture and the œsophagus is dilated. The walls of the dilated portion retain their normal thickness or are thinned. The stenosis is usually produced by hypertrophy of the muscular coat, by new growths, or by the cicatrization of ulcers and sloughs.

Spontaneous dilatation is produced by traction of the stomach downward from large herniæ; by habitual distension of the organ with food; by contusions causing paralysis. In lesser degrees, dilatation is produced by chronic gastritis and peritonitis.

In very rare cases, circumscribed, sacculated dilatations are produced by indigestible bodies, such as wood, metal, etc., in the stomach.

Diminution in the size of the stomach is produced by new growths, by hypertrophy of the muscular coat, by the cicatrization of ulcers and sloughs, and by habitual emptiness of the organ. In this way, the capacity of the stomach may become very small.

NEW GROWTHS.

Carcinoma occurs in the stomach in all its different varieties: medullary, scirrhus, epithelial, melanotic, and colloid.

It is a disease of advanced life, of equal frequency in men and women.

It is, as a rule, primary in the stomach, excepting the secondary nodules which may occur in the peritoneal coat, and belong rather to the peritoneum. But Cohnheim has described a case in which there were nodules in the wall of the stomach, secondary to carcinoma of the mamma.

It is usually situated at the pylorus and the lesser curvature; less frequently at other points. It may involve a small part, or nearly the entire wall of the stomach; often extends to the œsophagus, rarely to the duodenum. The new growth may take an annular shape, and form a ring around the stomach; this is especially the case at the pylorus; or it projects in the form of one or more flattened, broad tumors; or there is a more uniform thickening of the walls of the stomach for a considera-

ble distance ; or there are ulcers with thickened edges and ragged floors.

The disease begins in the submucous coat, or, according to some authors, in the gastric follicles. The mucous membrane is lifted up and pushed inward ; after a time, it usually ulcerates. The new growth may also extend outward, and invade all the other coats of the stomach. There may be also an hypertrophy of the muscular coat, either confined to the pylorus or involving the entire wall of the organ. The muscular coat then looks like a thick, gray layer, divided up by white bands. The gray tissue is smooth muscle ; the white bands are fibrous septa, which may become infiltrated with cells.

The post-mortem appearances vary according to the stage of the disease, and the way in which it has grown. At first, the new growth is confined to the submucous coat, and the mucous membrane is uniformly lifted up, or projects in flattened tumors. Very often, however, necrotic and ulcerative changes soon make their appearance. The mucous membrane ulcerates, and the new growth projects as a ragged, fungous mass ; or the new growth also ulcerates, and leaves a rounded ulcer with thickened edges ; or the new growth ulcerates, and at the same time goes on to invade the subjacent tissues. In this way, the wall of the stomach is perforated, and peritonitis ensues ; or adhesions are formed, and the perforation takes place into the intestines, the abdominal wall, or the diaphragm ; or the disease invades the neighboring organs ; the liver, pancreas, spleen, supra-renal capsules, kidneys, vertebræ, portal vein, and vena cava. The necrotic processes may even go on in the new growth in these other organs. Thus we find large cavities with fungous walls in the liver, communicating with the cavity of the stomach.

When the new growth is at the pylorus, it may seriously obstruct the egress of food, and in this way dilatation of the rest of the stomach may be produced.

Secondary tumors are very frequently found in the neighboring lymphatic glands, the liver, pancreas, lungs, and other viscera.

The above description applies to all the varieties of carcinoma, excepting the colloid form.

The distinction between the medullary and scirrhus forms seems to be unimportant. It depends merely on the relative proportion between the cells and stroma, a proportion which presents every sort of gradation. The presence of pigment also, forming melanotic cancer, seems to be merely an accidental complication. The structure of the medullary, scirrhus, and melanotic varieties is essentially the same: a fibrous stroma, inclosing cavities filled with cells; the cells polygonal, nucleated, and of an epithelial character.

Epithelial Cancer of the stomach consists of cells resembling the normal cylindrical epithelial cells of that organ. The cavities in which these cells are arranged are long, and resemble glandular follicles. The near relationship of the epithelial to the scirrhus and medullary cancer may be inferred from the fact that the secondary nodules in the liver are not always formed of cylindrical cells, but sometimes of polygonal cells. Epithelial Cancer with pavement cells is rare. It is secondary to similar growths in the upper part of the alimentary canal.

Colloid or alveolar cancer presents a different appearance. It usually forms a uniform infiltration of the wall of the stomach, especially of its pyloric portion. It has less tendency to ulcerate, but often invades the entire thickness of the wall of the stomach, and appears on its peritoneal surface in the form of irregular nodules. The disease may spread over a considerable portion of the peritoneum, but does not usually form secondary nodules. This form of cancer consists of a fibrous stroma, forming rounded alveoli. The alveoli are filled with a gelatinous material, in which are imbedded polygonal nucleated cells. The presence of the gelatinous material gives to the entire growth a peculiar soft consistence, and gray translucent color.

Sarcoma is found in the stomach in rare instances, both as a primary and a secondary growth. As a primary tumor, it is most frequent in young persons. It invades all the coats of the stomach, and projects inward as a fungous tumor.

Myoma occurs in the form of rounded tumors, which originate in the muscular coat, but gradually separate themselves from it, and project inward or outward. The submucous myomata are at first small tumors, lying loosely attached in the sub-

mucous tissue. As they grow larger, they push the mucous membrane inward, and appear as polypi, first with a broad, then later with a narrow pedicle.

The subserous myomata grow in the same way, and form rounded or polypoid tumors beneath the peritoneal coat. Sometimes these tumors are partly sarcomatous.

It seems probable that most of the fibrous tumors which have been described in this situation were myomata.

Lipoma grows in the submucous tissue, and forms rounded or polypoid tumors. They usually project inward, but sometimes outward beneath the peritoneum. They may also appear in the form of numerous small yellow nodules beneath the mucous membrane.

Fibroma papillare consists in an hypertrophy of the normal villi of the stomach, or a new growth of similar villi. This growth may appear as a uniform thickening of a considerable portion of the mucous membrane, or in the form of polypoid tumors. Such polypi are single or multiple. The smallest form little rounded prominences; the larger have a small pedicle and large body. Their surface is smooth or villous. They consist of a central portion, formed of connective tissue, fibres, and cells. This is covered with a layer of little villi, covered with epithelium, and sometimes containing follicular glands.

Adenoma is found in the shape of an hypertrophy of the glands of the stomach, which may assume a polypoid shape. Also, it occurs in the form of rounded tumors in the submucous coat, formed of glandular follicles, lined with cylindrical epithelium. Small glandular tumors, resembling the pancreas, have also been observed in the submucous and subserous coats.

Lymphatic New Growths of the stomach are seen in some cases of leucæmia. They form flattened or rounded tumors, 3-12" broad, of soft, white consistence, and resemble in their structure the lymphatic glands.

Cysts occur in the mucous membrane of the stomach from dilatation of the glandular follicles. They are usually small, but may reach a considerable size. They contain mucus and epithelium.

DEGENERATIONS.

Calcification of the mucous membrane is sometimes found as a metastatic deposit with extensive disease of the bones. The salts of lime are deposited in a granular form in the connective tissue between the glandular follicles, and more rarely in the muscular coat.

Waxy Degeneration sometimes invades the stomach. It attacks the vessels of the mucous membrane, and more rarely the cells of the glandular follicles. The mucous membrane appears thin and glistening.

THE INTESTINES.

MALFORMATIONS.

Diverticula of the intestines occur in several different ways :

(1.) The abdominal walls are cleft asunder at the navel. The ileum opens through this cleft by a narrow aperture in its wall. The lower portions of the ileum and the colon are small or entirely closed.

(2.) There is an opening in the abdominal wall as before, but there is not a direct opening into the ileum. There is a long diverticulum of the ileum, with an open end projecting into the opening in the abdominal wall.

(3.) The abdominal wall is closed. There is a diverticulum of the ileum connected with the navel by a solid cord.

(4.) There is an unattached diverticulum of the intestine. This is much the most common form. The diverticula occur only in the lower part of the ileum. They usually spring from the convex surface of the intestine, more rarely from its attached border. In the latter case, they are joined to the mesentery by a fold of peritoneum. The diverticulum forms a pouch, 1-6 inches long, of about the same diameter as the intestine, smallest at its free extremity.

Such diverticula never interfere with the functions of the intestines. They sometimes form part of a hernia. If they remain attached by a fibrous cord to the navel, this cord may be the cause of incarceration of a portion of the intestines.

Cloacæ consist in the union of the rectum, bladder, and organs of generation in a common outlet.

(1.) *Simple Cloacæ* are: (a.) Complete, and consist in the common opening of the urethra or ureters, the vagina and the rectum into the closed bladder, or into a sinus opening outward, which represents either the vagina or rectum. (b.) Incomplete. The rectum opens into the vagina, the bladder, or the urethra, while the lower part of the rectum is closed or absent.

(2.) *Cloacæ combined with cleft bladder.* (a.) The simple cleavage of the intestines is combined with cleft bladder. The anterior abdominal wall from the umbilicus to the symphysis, the symphysis, and the anterior wall of the bladder are absent; the gap is filled with a membrane which represents the posterior wall of the bladder. Into this membrane open the ileum, ureters, and vagina. (b.) The intestine is perfectly formed, but the rectum opens into a common sinus with the ureters and vagina; or the ureters open into the cleft bladder, and the rectum and external genitals are united; or the ureters open into the rectum, and the latter terminates normally.

(3.) *Cloacæ combined with abdominal hernia.* There is a hernial sac containing all the abdominal viscera. At the lower end of the sac is an opening leading into a sinus in which open the lower end of the ileum, the bladder or urethra, and the ureters. The rectum is absent.

Atresia Ani consists in a deficient development of the colon or rectum. The entire colon may be absent; the rectum may be absent, or represented by a solid cord; or the upper or lower part of the colon may be absent, or separated by a solid cord.

More rarely, blind terminations of the small intestines are found, and sometimes narrowing so complete as to close the canal.

The intestines are also found abnormally shortened in various degrees.

CHANGES IN POSITION.

Hernia.

The abdominal viscera may escape from the abdominal cavity through openings formed by arrest of development, or through

canals which are normally closed, or through thin portions of the abdominal wall. Herniæ, therefore, occur in a variety of situations.

(1.) *Hernia inguinalis*.—(a.) The hernia is congenital. The processus vaginalis of the peritoneum remains open, a portion of the intestines passes through into the scrotum, and is free in the cavity of the tunica vaginalis.

(b.) The hernia is acquired. It is oblique and formed by a projection of the intestines through the internal ring, inguinal canal, and external ring into the scrotum. The intestines push before them the peritoneum, which incloses them as their sac. Or it is direct, and the intestines pass through some part of the abdominal wall internal to the epigastric artery, and then through the external ring. They are covered with a sac of peritoneum.

(2.) *Hernia femoralis*.—The intestine, covered by a sac of peritoneum, passes through the femoral ring, descends along the crural canal in the inner compartment of the sheath of the femoral vessels as far as the saphenous opening; through this opening it escapes into the areolar tissue of the groin. The hernia may only pass into the femoral canal, and not escape through the saphenous opening; it may continue along the course of the vein beyond the saphenous opening; or it may project through several openings in the sheath of the vessels. This form of hernia is most frequent in the female. When incomplete, it not infrequently escapes observation during life.

(3.) *Hernia umbilicalis*.

(a.) *Congenital*.—In the foetal condition a portion of the ileum lies within the umbilical cord. If this coil of intestine does not pass back into the abdomen, and the regular closure of the umbilicus does not take place, there remains a sac, lined with peritoneum, filled with intestine, and from the end of the sac the umbilical cord is continued. The sac may contain only a small portion of intestine, or nearly all the abdominal viscera. The wall of the sac is thin, and may rupture before or at the time of birth. In other cases, either spontaneously or by surgical interference, the sac is obliterated and the hernia reduced.

(b.) *Acquired*.—The intestine covered by a sac of peritoneum projects into a sac formed by the skin at the umbilicus. This hernia occurs in young infants when the navel is not firmly cicatrized; in adults, when there has been a small hernia in infancy; and when the navel has been dilated by pregnancy, etc.

(4.) *Hernia ventralis*.—There may be herniæ of the abdominal wall in the median line or to one side of it. The intestine may project through some defect in the fasciæ, or there may be thinning and pouching of all the coats of the abdomen at some point.

(5.) *Hernia ischiadica*.—The intestine, covered by a sac of peritoneum, passes along the course of the sciatic nerve through the sacro-sciatic foramen under the glutæus maximus muscle. It is very rare.

(6.) *Hernia foraminis ovalis*.—The intestine, covered by a sac of peritoneum, passes on the inner side of the obturator vessels through the obturator foramen.

(7.) *Hernia perinæalis*.—The intestine, covered by peritoneum, passes between the fibres of the levator ani muscle and projects into the space between the rectum, the tuberosity of the ischium, and the point of the sacrum.

(8.) *Hernia vaginalis*.—The intestine, covered by peritoneum, passes downward between the rectum and vagina, and presses the posterior wall of the vagina outward through the vulva. If the hernia is large, the wall of the vagina drags down the posterior lip of the cervix and the body of the uterus.

(9.) *Hernia rectalis*.—The hernia takes place between the bladder or vagina and the rectum. The wall of the rectum may then be pressed outward through the anus.

(10.) *Hernia diaphragmatica*.

(a.) *Congenital*.—This is produced by defective development of the diaphragm, which may be partly or entirely wanting on one side. Part of the intestines or most of the abdominal viscera may thus pass into a pleuritic cavity. They are covered by a sac formed of peritoneum and pleura, or are without any sac. Or the diaphragm is complete, but part of it is pouched into the pleural cavity. In rare instances, persons with this lesion reach adult life.

(b.) *Acquired*.—This is produced by wounds or ruptures of the diaphragm, or the hernia projects through one of the natural openings of the diaphragm, or through an opening formed by a fat hernia.

(11.) *Hernia Retroperitonæalis*.—The intestine passes into the fossa jejuno-duodenalis. It consists of a small part of the jejunum, or of most of the small intestines. It is covered with a duplicature of peritoneum from the posterior abdominal wall.

For a detailed account of hernia, the text-books on surgery should be consulted.

INCARCERATION.

(1.) The most common form is that in which a portion of intestine is strangulated by a fibrous band. Such fibrous bands are produced by peritonitis, or are remains of foetal growth. They pass from the intestines to the abdominal wall, or from one part of the intestines to another. The intestine becomes in some way caught under one of these bands, and is compressed by it. The stricture thus produced may cause a gradual accumulation of feces in the intestine above it, and may last for a long time before death ensues. In other cases, the stricture interferes at once with the circulation of the blood; the intestine is intensely congested, becomes gangrenous, and death takes place with the symptoms of general peritonitis.

(2.) A portion of intestine becomes caught in some abnormal opening in the mesentery or omentum, or in the foramen of Winslow, or between the two layers of the mesentery.

(3.) A coil of intestine makes half a turn at its base, so that the two sides of the loops cross at its base. In this way, the lumen of the intestine is completely closed, and the vessels are compressed, so that congestion, peritonitis, and gangrene result. This form of incarceration is most frequent in the ascending colon. In the small intestine it only occurs when the gut is fixed by old adhesions.

(4.) A portion of the intestine, with its mesentery, makes one or more complete turns on itself, closing the canal, and compressing the vessels.

(5.) A portion of the intestine makes a half or entire turn

about its long axis. This is very rare, and only occurs in the colon.

(6.) The mesentery of a part of the intestine is long and loose, in consequence of a dragging down of the intestine by a hernia or by habitual constipation. The portion of intestine thus permitted to hang down is habitually filled with feces, and by its pressure on some other part of the intestine produces an incomplete stricture.

INTUSSUSCEPTION.

This change of position consists in the invagination of one portion of intestine in another portion. Usually this takes place in the direction of the peristaltic movements, from above downward; more rarely, in the opposite direction.

The parts are found in the following condition: There are three portions of intestine, one within the other. The inner portion is continuous with the intestines above the intussusception; its peritoneal coat faces outward. The outer portion is continuous with the intestine below; its peritoneal coat also faces outward. The ~~inner~~ *middle* portion is turned inside out, its mucous membrane is in contact with the mucous membrane of the outer portion. In rare cases, the intussusception is complicated by the invagination of a second portion of intestine in the inner tube, and even by a third intussusception into the second one. These changes occur both in the large and small intestine; most frequently the lower part of the ileum is invaginated in the colon. The invaginated portion may be from a few inches to several feet in length. The lesion is most frequently found in early childhood.

The intussusception, by the dragging and folding of the mesentery which it produces, causes an intense congestion of the parts, and even large hæmorrhages between the coats of the intestine. The congestion may induce fatal peritonitis, or gangrene of the intestine, or chronic inflammation and adhesions, and the patient lives for a considerable time with symptoms of stricture. In other cases, the invaginated portion of intestine sloughs, the outer and inner portion become adherent, and the patient recovers, with or without some degree of stricture.

Besides this grave form of intussusception, we often find, especially in children, one or more small invaginations not attended with congestion or inflammation. These are formed during the death-agony or immediately after death.

PROLAPSUS.

Prolapsus of the rectum consists in the protrusion of the lower part of the rectum through the anus. It begins as a slight prolapse of a small part of the rectum, or of its mucous membrane, only occurring in the act of defecation. The prolapsed portion becomes gradually larger and larger; it no longer returns of itself; it is constricted by the sphincter, becomes hyperæmic, swollen, and inflamed, so as to form a mass of large size.

The lesser degrees of prolapse are common, especially in children, and are of little importance. The higher degrees may become the seat of severe inflammation and ulceration, and, in rare cases, gangrene and death may supervene.

Sometimes only the mucous membrane prolapses, and the prolapse may not involve the entire circumference of the rectum, but only one or more folds of the mucous membrane.

TRANSPPOSITION.

The position of the intestines may be the opposite of that usually found. The transposition may affect all the abdominal viscera; the liver and cæcum are on the left side, the stomach and spleen on the right side; or only a single viscus is transposed.

WOUNDS, RUPTURE.

Penetrating wounds of the intestine usually prove rapidly fatal from the peritonitis which supervenes. Sometimes, however, the wound is closed by the formation of adhesions with the neighboring parts. The intestine may then remain bound down by adhesions, or after a time become free. Sometimes the wound in the intestine becomes adherent at the position of the wound of the abdominal wall, and an intestinal fistula is formed.

Rupture of the intestines is produced by severe contusions. It is noticeable that contusions sufficiently severe to produce large ruptures of the intestines may not leave any bruises or marks on the external surface of the body, even when the patient lives for several days after the receipt of the injury.

Perforation of the intestines will be considered under the head of ulcers.

INFLAMMATION.

The Rectum.—Catarrhal inflammation of the rectum is acute or chronic.

In acute inflammation, we find the intestine congested, swollen, covered with a thin layer of mucus, pus, and epithelium; the epithelium in places destroyed so as to form superficial ulcers; the solitary glands enlarged. In chronic inflammation, the mucous membrane is congested, or anæmic, or mottled with black patches. There are ulcers, either superficial or deep, and even perforating, so as to form fistulous openings into the surrounding tissues. Other parts of the mucous membrane hypertrophy, either around a solitary follicle, or around a group of straight follicles, or by cell-growth beneath the mucous membrane, and project in the form of polypi. In other cases, the mucous membrane is uniformly thickened. The muscular coat is relaxed and thinned, or, more frequently, hypertrophied. This hypertrophy is frequently associated with thickening of the connective tissue around the rectum, and may cause fatal strictures.

Croupous inflammation of the rectum occurs with acute dysentery and after the injection of irritating substances. The mucous membrane and submucous tissue are congested, swollen, and infiltrated with serum. Its surface is covered with blood, epithelium, mucus, and a thin, brownish, membranous layer, composed of epithelium mixed with coagulated fibrine. This false membrane may be in patches, or may form a complete cast of the rectum. In more severe cases, the entire thickness of the mucous membrane is infiltrated with fibrine, so that large patches become gangrenous and are cast off.

In this way, the greater part of the mucous membrane may be destroyed. The milder forms of croupous inflammation, in which only the superficial layers of the mucous membrane are destroyed, leave no permanent lesions. The deeper sloughing and ulcerative processes, on the other hand, leave large cicatrices and contractions of the intestines.

The connective tissue around the rectum frequently shares in the inflammatory changes of the intestine, and becomes indurated. It may also be the seat of idiopathic inflammatory changes, producing induration or abscesses around the rectum.

The Colon.—The entire length of the colon is subject to the same inflammatory changes as the rectum. Acute and chronic catarrhal inflammation, with ulceration; thickening of the muscular coat and the formation of mucous polypi; croupous inflammation, with the formation of large ulcers, both occur. In rare cases, idiopathic croupous inflammation of the colon occurs, and causes death.

The Cæcum.—Catarrhal inflammation of the cæcum, or typhlitis, is not uncommon. It is usually produced by an habitual accumulation of fæces in this portion of the intestines. The course of the disease is usually chronic, but marked by acute exacerbations. The mucous membrane is at first congested, afterward ulcerated; there is a slow, suppurative inflammation of all the coats of the intestine and of the surrounding tissues; fistulous openings are formed through the wall of the cæcum, through which the fæces pass into the cavity of the abdomen, and cause fatal peritonitis, or produce abscesses which are shut in by adhesions; or the fæces pass through the peritoneum into the muscles of the posterior wall of the abdomen, and form large cavities filled with pus and fæces.

Perityphlitis.—There may be an idiopathic inflammation of the subperitoneal tissue just beneath the cæcum. It forms large abscesses, which may perforate the cæcum and allow the fæces to escape. Or the wall of the cæcum may become relaxed, so that a persistent and even fatal constipation is produced.

The Appendix Vermiformis is sometimes the seat of catarrhal inflammation and ulceration, excited by the presence in it

of small foreign bodies or hardened fæces. The ulcers frequently cause perforation, and, in consequence, the formation of abscesses, adhesions, and peritonitis.

In other cases, the inflammation leads to closure of the neck of the appendix. It may then, by the accumulation of mucous and serum in its cavity, become a cyst of considerable size.

The abscesses which are formed in consequence of typhlitis, perityphlitis, or perforation of the appendix, may perforate into the bladder, vagina, uterus, acetabulum, ascending vena cava, other parts of the intestinal canal, through the abdominal wall into the muscles of the abdominal wall and the pelvis, through the diaphragm into the pleural and pericardial cavities.

Ileum.—The ileum, especially its lower portion, is often the seat of acute and chronic catarrhal inflammation. In acute inflammation, the mucous membrane is congested and swollen, the solitary and agminated glands enlarged.

In chronic inflammation, both the mucous and muscular coats are thickened, the mucous membrane is congested and anæmic or pigmented, often ulcerated.

Croupous inflammation occurs in children in the form of aphthæ; in adults, usually as the result of irritant poisons. I have seen, however, idiopathic croupous inflammation of the ileum in a few cases. The inflammation was most intense over about two feet of the intestine, but extended in a lesser degree down to the end of the ileum. Where the inflammation was most intense, the mucous membrane was congested, covered with a layer of fibrinous exudation, the solitary and agminated glands were not enlarged; there was an infiltration of lymphoid cells and fibrine through the entire wall of the intestine, and a recent fibrinous exudation on its peritoneal surface, which did not, however, extend to a general peritonitis.

Duodenum.—Catarrhal inflammation of the duodenum occurs by itself or with gastritis. It runs a chronic or acute course. It is principally important from its effect on the gall-duct, obstructing the passage of bile into the intestine.

Suppurative Inflammation of the submucous coat of the intestines is said to occur in rare cases. It is usually metastatic. It takes place in the form of foci of varying extent, which perforate either inward or outward.

ULCERATION.

1. *Catarrhal Ulceration* occurs in the colon and ileum, with catarrhal inflammation of the mucous membrane. Small rounded patches of the mucous membrane appear at first deeply injected, then soften and fall off, leaving ulcers which are at first superficial and then deep. These ulcers then spread laterally, or several become joined, so as to form large ulcers, of which the edges are formed by the thickened mucous membrane, their floor by the muscular or peritoneal coat, infiltrated with cells. Such ulcers may destroy a large part of the mucous membrane of the large intestine, and may perforate. They cicatrize in the form of broad, hard, pigmented patches. By their contraction and the accompanying hypertrophy of the muscular coat, they may produce strictures.

2. *Follicular Ulcers* are formed by the inflammation of the solitary glands in the large intestine, and of the solitary and agminated glands in the small intestine. These glands increase in size; the mucous membrane over them softens and falls off so that they project as little, soft, rounded bodies, filled with a whitish puriform material. The gland then ruptures, discharges its contents, and thus forms a rounded ulcer with overhanging edges. In the agminated glands, the process is the same on a larger scale. When the ulcer is thus formed, its base and edges become infiltrated with lymphoid cells, and in this way the ulcer may increase in breadth and depth, and even perforate the peritoneal coat.

The ulcers which are frequently called tuberculous are really follicular ulcers; but miliary tubercles may form in their edges and near them as a secondary process.

3. *Croupous Ulceration* occurs in the large intestine from the infiltration and sloughing of portions of the mucous membrane.

4. *Simple perforating Ulcers* occur in the duodenum. They resemble exactly the perforating ulcers of the stomach, and seem to be formed in the same way. They are found with ulcers of the stomach, or alone. They may be single or multiple.

5. *Syphilitic Ulcers* are found in the rectum as an extension

of chancres of the vulva and perineum. They may lead to induration and stenosis of the rectum.

EMBOLI.

Emboli have been observed in the superior mesenteric artery in a number of instances; in the inferior mesenteric they are less frequent. They produce an intense congestion of the intestine, usually with hæmorrhage from the bowels during life. The intestine appears of a livid red color, swollen, moist, sometimes almost gangrenous.

STENOSIS AND DILATATION.

Stenosis of the intestine is produced by hypertrophy of the muscular coat, by cicatrices, by new growths, by the pressure of tumors, (in one case by the pressure of an anterior spina bifida on the rectum,) by peritoneal adhesions, by foreign bodies.

Dilatation of the intestine is produced by the accumulation of fæces and gas above a stricture, or by relaxation of the walls of the intestine from concussion, habitual repletion, peritonitis, rheumatism, typhus, dysentery, cholera, over-stimulation by injections, and purgatives, or from some disease of the nervous centres. In some cases, not only the calibre, but also the length of the intestine is increased.

False Diverticula are formed by a hernial protrusion of the mucous and peritoneal coats. They form multiple nipple-shaped pouches, from the size of a pea to that of a walnut. They are usually on the convex side of the intestine, less frequently on its concave side. They are found at all parts of the large and small intestine. They usually cause no symptoms during life. In one case, however, they appeared to be the cause of an acute peritonitis, though no perforation was discovered.

Dilatation of the Appendix Vermiformis is produced by the stenosis or occlusion of its canal at some point. Beyond the point of obstruction the canal dilates so as to form a large sac with thin walls. The contents of the sac are thick mucus and epithelium, or serum.

NEW GROWTHS.

Myoma.—Tumors composed of smooth muscle and fibrous tissue are found as small, rounded, hard bodies. They grow from the muscular coat, but soon project into the submucous coat, and push inward the mucous membrane. They may entirely obstruct the cavity of the intestine, and may then be seized by the peristaltic action of the intestine, and produce an intussusception. In the duodenum such tumors may obstruct the opening of the biliary duct. Such tumors are also said to project under the peritoneal coat.

Lipoma may occur at any part of the intestinal tract. The tumors project beneath the mucous coat, and form polypi, sometimes of considerable length; or they are simply hypertrophies of the normal appendices epiploicæ. In these latter, there may be an increase of fibrous tissue and sclerosis; or the fat may become fluid, and form a sort of cyst; or they may calcify. Such tumors sometimes, by the atrophy of their pedicles, become free in the abdominal cavity.

Adenoma occurs usually in the form of polypoid tumors. They are found in the rectum, and are most frequent in children. They are single or multiple, small or large, rounded or polypoid. They consist of long glandular follicles, lined with cylindrical epithelium, and contained in a fibro-cellular stroma.

Fibroma papillare has already been mentioned as produced by chronic inflammation of the intestines. The tumors are of polypoid shape, and composed of fibro-cellular tissue, covered with epithelium. They are often made up of a great number of tufts, each covered with epithelium.

Angioma is of very rare occurrence in the intestines, except in the form of the hæmorrhoids of the rectum, and as a degeneration of the polypi mentioned above.

Hæmorrhoids are a form of angioma. They are produced by the dilatation of the veins supplying the lower part of the rectum. These veins communicate directly with the ascending vena cava through the internal ilia; but they also have large anastomoses, with the inferior mesenteric, and in this way they communicate with the portal system. Hæmorrhoids are inter-

nal or submucous; external or subcutaneous; and mixed, or partly submucous and partly subcutaneous. The dilatation of the veins is accompanied with growth of connective tissue about them. The blood in the dilated veins sometimes coagulates and forms thrombi.

Carcinoma of the intestines is usually primary. It may occur as medullary, scirrhus, colloid, or cylindrical-celled epithelial cancer. At the anus, flat-celled epithelial cancer also occurs. The most frequent form is the epithelial cancer with cylindrical cells. The rectum and colon are the parts most frequently attacked. The new growth usually surrounds the intestine, and produces fatal strictures. In other cases, it perforates into the abdominal cavity.

Sarcoma, both white and pigmented, has been observed in the rectum.

Lymphoma occurs in the intestines in cases of leucæmia. The nodules are soft, white, flattened, or rounded, and may ulcerate. They are formed either as an hypertrophy of the solitary and agminated glands, or as growths, where these glands are not normally found.

Cysts are of rare occurrence. They are found in some of the polypoid growths. Rokitsky describes cysts in the intestines and mesentery, which were formed by cystic degeneration of the chyle glands.

Tubercles of the intestines are principally confined to the serous coat and to the walls of ulcers. The so-called tubercular ulcers of the mucous coat are formed by the enlargement and ulceration of the solitary and agminated glands. They differ in no respect from the ordinary follicular ulcers already described. Such follicular ulcers, when of long standing, may at their bases be infiltrated with small cells; the lymphatics over them appear as white streaks, and in the peritoneal coat there may be a collection of miliary tubercles.

DEGENERATION.

Calcification attacks, in rare cases, the wall of the intestines, appearing in fibrous thickenings of the peritoneal coat.

Waxy Degeneration may affect the blood-vessels of the mu-

cous membrane and of the villi; the villi themselves, and, more rarely, the muscular coat.

ABNORMAL CONTENTS.

The ordinary faecal contents of the small and large intestines are, in inflammation, mixed with serum, epithelium, mucus, and blood.

Blood in the intestines, from whatever source it is derived, when recent, is still red and coagulated. After a longer stay in the intestines, it becomes converted into a black, granular mass.

Concretions are formed either of hard, dry, faecal masses, or of mucus mixed with dried faeces, or collected around a foreign body, and impregnated with salts of lime. Such concretions are usually small, but may reach the size of a man's fist. They are round, oval, laminated, angular, or of irregular shape. They are found to consist of mucus, bile, faecal matter, carbonate and phosphate of lime, and triple phosphate. They may give rise to no evil effects, or may cause inflammation, ulceration, and perforation, especially of the appendix vermiformis. Sometimes the ulcerative process stops, and the concretion is left attached to the mucous membrane by membranous adhesions.

Foreign bodies of every variety may be swallowed, and produce different effects in the intestines, according to their nature.

PARASITES.

Ascaris lumbricoides is found in the small intestine, either singly or in numbers. In rare cases, a number of worms may form a mass, which produces inflammation, ulceration, and perforation.

Oxyuris vermicularis is found in large numbers in the rectum.

Tricocephalus dispar is found in the caecum.

Ankylostomum duodenale is found in the duodenum, and may give rise to considerable hæmorrhages.

Trichina spiralis is found in its adult condition in the small intestine.

Pentastomum denticulatum occurs in the submucous tissue of the small intestine in an encapsuled condition.

Cysticercus cellulosæ has been seen in a few cases on the mucous membrane.

Tenia solium, *Tenia mediocanellata*, and *Bothriocephalus latus*, are all found in the small intestine.

THE PERITONEUM.

MALFORMATIONS.

Arrest of development in the peritoneal sac occurs in the shape of fissures in the mesial line, or external to it; in the case of the diaphragm being absent, of a fusion with the pleura; as defective development of the mesentery, the omentum, and the other folds of the peritoneum.

Excess of development occurs in the shape of unusual length of the mesentery, the omentum, and the other fold of the peritoneum; or of supernumerary folds and peritoneal pouches. These are chiefly found in the hypogastric, and, more especially, in the iliac and inguinal regions, and near the fundus vesicæ. There is access to these sacs by a well-defined fissure or ring, which is frequently surrounded by a tendinous band lying in the duplicature. They may give rise to internal incarceration of the intestines. (Rokitansky.)

HYPERÆMIA AND HÆMORRHAGE.

Congestion of the peritoneum is general or partial. It is caused by obstructions to the venous circulation in the portal vein and vena cava, and by inflammation. It produces serous effusions in the peritoneal cavity, thickening of the peritoneum, small extravasations, and consequent pigmentation of the peritoneum.

Extravasations in and beneath the peritoneum are found with purpura, scurvy, after contusions, parturition, etc.

Hæmorrhage into the peritoneal cavity is produced by wounds, contusions, rupture of the abdominal viscera, and of aneurisms.

INFLAMMATION.

Inflammation of the peritoneum is acute or chronic, general or partial.

Acute General Peritonitis produces congestion, and an exudation composed of fibrine, lymphoid cells, and serum. The amount of congestion is variable, and does not always remain visible after death. It is most intense at the point where the inflammation begins, especially if this is produced by a wound, or by perforation of the intestines, or of an abscess. That congestion exists during life, can be seen in animals in whom peritonitis is artificially excited. The stage of congestion may exist for a certain time before exudation takes place. How long, and under what conditions this is the case in the human subject, is not known. There are cases of puerperal women who manifest all the symptoms of peritonitis, and die from it, without any perceptible lesions of the peritoneum or uterus after death. In such cases, we may admit the existence of a peritonitis without exudation, and in which the congestion has disappeared after death. The exudation, when present, is scanty or abundant. It forms a thin, gray membranous layer, agglutinating neighboring parts; or the peritoneum is coated with thick, yellow material, composed of fibrine and lymphoid cells; or there is a large amount of yellow, purulent serum in the abdominal cavity. Sometimes there are little extravasations of blood in the tissue of the peritoneum, and mixed with the exudation, and sometimes the purulent serum is of a peculiar dirty brown, fetid character. The muscular coat of the intestines may be infiltrated with serum, relaxed and paralyzed. General acute peritonitis is usually fatal, but may pass into the chronic form, or may disappear, leaving membranous adhesions. In some cases, these adhesions will inclose masses of pus and fibrine. These masses may then become dry, cheesy, or even calcified, or they become fluid and purulent. Such abscesses inclosed by adhesions may rupture into the peritoneum, and cause a second peritonitis, or they may cause a general chronic peritonitis, or perforate the intestines, or lead to

the formation of large abscesses beneath the peritoneum, or perforate through the skin.

Peritonitis is idiopathic, secondary, or metastatic.

Idiopathic peritonitis is usually produced by wounds and contusions, or occurs in connection with the puerperal condition in women; except in such cases, it is rare.

Secondary peritonitis is produced by the continuation of inflammatory and gangrenous processes of the abdominal viscera and wall. Thus, in the stomach and intestines, we find perforating ulcers, irritative poisons, malignant new growths, incarceration and intussusception, retention of fæces, and, rarely, severe catarrhal and croupous inflammation, producing a local peritonitis. Lesions of the uterus, the ovaries, the fallopian tubes, frequently cause general peritonitis. Inflammation of the urinary bladder, vesical and renal calculi, rupture and perforation of the bladder, malignant growths of the bladder, nephritis, perinephritis, and tuberculosis of the urinary organs, may all cause peritonitis. In the liver we find abscesses, biliary calculi, new growths, hydatid cysts, and thrombosis of the veins acting as irritating causes.

In the spleen and pancreas, inflammation, suppuration, infarctions, and new growths; in the cellular tissue, beneath the peritoneum, and in the lymphatic glands, inflammation and suppuration, and new growths; in the abdominal vessels, ruptures of aneurisms, thrombosis of veins, may excite peritonitis. In the abdominal wall, inflammation, suppuration, and new growths; in the diaphragm, perforation from empyema and abscesses of the lungs often cause peritonitis. In some cases, without perforation, there is at the same time pleurisy and peritonitis, and it is difficult to say whether the two are coincident, or whether one is secondary to the other.

In the bones and muscles, caries of the vertebræ and ribs, with abscesses in the muscles, may cause peritonitis.

In the peritoneum itself, new growths and hydatid cysts may cause inflammation.

Metastatic peritonitis is found with pyæmia, the acute exanthemata, scurvy, and Bright's disease.

Partial acute peritonitis is usually produced by inflammation

of some of the viscera which the peritoneum covers. The spleen, the liver, and the uterus are very often found covered with adhesions produced by attacks of local peritonitis. Such a partial peritonitis may give rise to few or no symptoms during life, and usually subsides, leaving membranous adhesions. Sometimes, however, it produces abscesses in the subperitoneal tissue.

In rare cases, the omentum alone is the seat of acute inflammations, sometimes leading to purulent infiltration.

General chronic peritonitis is usually the result of an acute peritonitis. This is especially the case when masses of exudation are shut in by adhesions, and give rise to a series of chronic inflammatory processes.

In other cases, a long-continued obstruction to the portal circulation, caused by disease of the liver and heart, produces a thickening of the peritoneum and membranous adhesions between the viscera. There is usually at the same time ascites. In these cases the inner surface of the peritoneum is often covered with numberless little granules formed of clumps of epithelial cells.

In rare cases, the exudation has a gelatinous appearance, and is produced in enormous quantities, so that the abdomen is distended and the viscera compressed.

In other cases of chronic peritonitis, there is a very considerable thickening of the peritoneum with thick, lardaceous, false membranes, which cover the parietal peritoneum, form adhesions between it and the viscera, and cause the intestines to adhere together, so that they form a solid tumor. The adhesions are composed of connective tissue, fibrine, and fat, are yellow, of waxy consistence, and usually mottled with small hæmorrhages. The lumen of the intestines is contracted, the peritoneal and muscular coats thickened.

In tubercular peritonitis there are found numbers of gray, miliary tubercles in the peritoneum and in the false membranes. The proportion between the number of tubercles and the amount of false membranes varies very much in different cases; some serous effusion is also usually present.

Chronic partial peritonitis is more common, and usually de-

pend on lesions of the viscera. The spleen, liver, and uterus are frequently the seat of a chronic inflammation, producing thickening of their capsules and membranous adhesions. Ulceration of the intestines, typhlitis, subperitoneal abscesses, etc., all lead to chronic partial peritonitis. In rare cases, the omentum alone is the seat of a chronic inflammation, producing fibrous thickening and adhesions.

The most important feature of such a partial peritonitis is the interference with, or even stricture of, the intestine produced by the adhesions.

The subperitoneal tissues sometimes become the seat of suppurative inflammation, by which abscesses are formed. These retroperitoneal abscesses are caused by perinephritis, pericystitis, perityphlitis, periproctitis, perimetritis, periovaritis, inflammation of the muscles of the back and abdomen, caries of the vertebrae, etc. The abscesses usually reach a considerable size, they invade the neighboring soft parts, produce caries of the bones, and finally perforate inward through the peritoneum, or outward through the skin.

GANGRENE.

Gangrene of the peritoneum is produced by incarceration and intussusception of the intestines, by abscesses, and, in infants, by gangrene of the umbilicus. The portion of peritoneum affected appears as a slough surrounded by a zone of intense congestion, or as a softened, black, putrid mass.

ASCITES.

Besides the turbid fluid, composed of serum, pus, and fibrine, which we find as an inflammatory exudation in the peritoneal sac, we also meet with simple dropsical accumulations of serum.

The serum, in these cases, is nearly colorless, yellow, green, or red. It is perfectly transparent, or opalescent, or contains fibrinogenic material, which coagulates when exposed to the air. The composition of the fluid resembles that of the serum of the blood.

The peritoneum appears unchanged, or is white and thick, or there may be adhesions between the different viscera.

The fluid is usually free in the peritoneal cavity, but may be sacculated by adhesions. Sometimes, especially in children, there is œdema of the omentum alone.

The serum not only distends the abdominal walls, but pushes up the liver and diaphragm, and causes the lower ribs to be bulged outward.

A small amount of ascites is found often in cachectic and ill-nourished persons. A larger amount is usually caused by diseases of the heart, kidneys, liver, spleen, lungs, and by any obstruction to the portal vein or ascending vena cava.

Air is sometimes found in the peritoneal sac after perforation of the stomach, intestines, uterus, and abdominal wall. The spontaneous formation of gas in the peritoneum is doubtful.

NEW GROWTHS.

Fibroma of the peritoneum is rare. The tumors grow from the subserous tissue. Those which have been described in this situation on the intestines are probably mostly myomata. Others, however, grow beneath the parietal peritoneum, project outward into the muscles, and are true fibromata.

Lipoma.—An abnormal accumulation of fat in the omentum and mesentery, and beneath the peritoneum, is often seen. Besides this, circumscribed fatty tumors are found beneath the intestinal and parietal peritoneum. On the intestines they are usually hypertrophies of the appendices epiploicæ. These tumors often degenerate, calcify, become indurated, their pedicle atrophies, and they become free in the abdominal cavity.

When they grow beneath the parietal peritoneum, they may form fat herniæ, hernia lipomatosa. At the umbilicus, in the inguinal canal, and along the vas deferens, in the crural ring, and in the foramen obturatorium, fatty tumors may grow, project outward under the skin like herniæ, and, by drawing the peritoneum after them into a pouch, may lay the foundation of a future intestinal hernia.

Myxoma.—In persons much emaciated and debilitated by chronic disease, the fat beneath the intestinal and parietal peritoneum sometimes undergoes mucous degeneration, and is converted into a gelatinous substance of a light yellow color.

In the pelvis of the kidneys, new growths of mucous tissue are found, which project inward into the abdominal cavity. Such tumors, however, are usually combinations of myxoma and sarcoma.

Sarcoma appears in the form of solitary, slowly-growing tumors behind the peritoneum, or between the folds of the mesentery. They may reach a large size, and by their pressure on the veins and the intestines, cause thrombosis of the large veins and obstruction of the intestines. They may give rise to metastatic tumors of the peritoneum and of the viscera.

In the peritoneum itself, especially in the omentum, the mesentery and the appendices epiploicæ sarcomata are found both as primary and secondary tumors. They are frequently very vascular.

Carcinoma of the peritoneum is either primary or secondary. It assumes the form of colloid or of true carcinoma, either hard or soft.

The colloid form, when primary, involves the greater part of the peritoneum, and forms a large mass which distends the abdomen. The omentum is changed into a large gelatinous mass; the subjacent muscles, the lymphatic glands, and the liver are infiltrated with the new growth, and soft gelatinous masses project into the peritoneal cavity. The umbilicus is sometimes invaded, so as to project outward in the form of a semi-transparent tumor. The appearance of the new growth is that of a soft, jelly-like mass, imbedded in a fibrous stroma. The minute structure consists of fibrous alveoli, containing epithelial cells, and a homogeneous, transparent, gelatinous substance. When the disease is far advanced, no cells are to be found.

When the new growth is secondary, it appears in the form of numerous little nodules on the free surface of the peritoneum.

True carcinoma is either primary or secondary. It usually appears in the form of numerous small nodules in the intestinal and parietal peritoneum. These nodules are small, firm, and white. They are often united by a flat layer of the same new growth, so that the peritoneum is changed into a uniform, thick, white membrane. Less frequently, carcinoma appears as solitary, soft, medullary growths.

Tubercles.—Gray and yellow miliary granulations are found in the peritoneum in some cases of general tuberculosis. They are found in the omentum without any inflammatory lesions, or in the peritoneum, over ulcers of the intestines, or in connection with fibrinous exudation and membranous adhesions in tubercular peritonitis.

Cysts.—In rare cases, dermoid cysts containing fat, hair, bones, etc., are found in the parietal and visceral peritoneum, and in the omentum. These are probably congenital. Rokitsky describes a cystic degeneration of the mesenteric glands. In the broad ligaments of the uterus, both serous and colloid cysts are found.

PARASITES.

Echinococci are found in hydatid cysts, beneath the capsule of the liver, in the omentum, and beneath the parietal and visceral peritoneum. They are surrounded by false membranes.

THE LIVER.

MALFORMATIONS.

Congenital malformations of the liver are not common, and are of little practical importance. The organ may be entirely wanting, the lobes may be diminished or increased in number; its form may be altered, so that it is rounded, flattened, triangular, or quadrangular. The gall-bladder may be wanting, the ductus choledochus may be double, both ducts emptying into the duodenum, or one emptying into the duodenum, the other into the stomach. The single ductus choledochus may also empty into the stomach.

In congenital transposition of the viscera, the liver is found on the left side, the stomach and spleen on the right side.

Small, isolated bodies, having the same structure as the liver, have been found in the suspensory ligament. They are analogous to the little accessory spleens.

CHANGES IN SIZE AND POSITION.

Changes in the size and form of the liver are frequent. The congenital malformations remain during life, and the liver is found quadrangular, or with abnormal fissures and lobes, or with abnormal size and shape of the lobes. The left lobe especially varies very much in size.

In females we find changes in the shape of the liver produced by tight lacing. By the narrowing of the base of the thorax, the organ is compressed from side to side, and its convex surface is pressed against the ribs. In consequence of this, there are found ridges and furrows on its convex surface. In consequence also of the circular constriction, a part of the right, and usually of the left, lobe also becomes separated by a depression. Over this depressed and thinned portion of the liver, the capsule is thick and opaque. In extreme cases, the depressing and thinning reach such an extent that there is only a loose, ligamentous connection between the separated portion and the liver.

New growths, hydatid cysts, and abscesses often produce a marked increase in the size of the liver.

Fatty and waxy degeneration increase the size of the liver.

Cirrhosis sometimes increases the size of the liver, but more commonly diminishes it, and produces lobulation. Syphilitic hepatitis, obstruction of branches of the portal vein, and partial inflammations and cicatrizations, also produce lobulation of the liver.

Changes in the position of the liver are produced by alterations in its size, by pressure downward from the thoracic cavity, and upward from the abdomen, by the constriction of tight lacing, by tumors or circumscribed serous exudation between the liver and diaphragm, by curvature of the spine.

When the liver or lower portion of the liver is constricted by tight lacing, the axis of the organ falls downward, and the edge of the right lobe protrudes far below the margin of the ribs. If there is at the same time a deep depression in the right lobe, from the same cause, and the intestines lie in this groove, it may seem as if there was a tumor, separated from the liver, in the right iliac region. When the upper third of the liver is com-

pressed, its axis is carried downward, and the entire organ pushed toward the median line. The liver may then fill the upper half of the abdominal cavity.

When the contents of the thorax are increased, the liver is pushed downward. In pulmonary emphysema, the liver may be depressed from one to one and a half intercostal spaces.

In pleurisy, empyema, and hydropneumothorax of the right side, the liver is pushed downward and dislocated, so that its convex surface is in apposition with the abdominal wall. When the effusion is in the left pleural cavity, the liver is pushed toward the right side, and slightly depressed.

Very great serous effusion in the pericardium may depress the liver, especially the left lobe.

Intra-thoracic tumors depress the liver, according to their situation.

When there is fluid or a new growth in the abdomen, or gas in the intestines, the liver is pushed directly upward, without any change in its axis.

Structural changes in the liver increase or diminish its size, and cause an increase or diminution in its thickness.

There have been a very small number (3) of cases reported of dislocated and movable liver. They all occurred in women who had borne children. The diagnosis was made during life, and no post-mortem examination of the cases was made.

The transverse colon may become fixed above the liver, so as to push it backward, downward, and to the right.

INJURIES.

Wounds of the liver produce hæmorrhage and inflammation. They are usually fatal; but the patient may recover, even after the destruction of a considerable portion of the organ.

Rupture of the liver is produced by severe contusions and falls; it results in large hæmorrhages, and is very fatal.

HYPERÆMIA.

Hyperæmia of the liver is either an active or a passive process.

In health, the amount of blood in the liver varies at different

times, being regularly increased during the process of digestion. When the digestive process is irritated by the ingestion of spirits, spices, etc., the hyperæmia assumes abnormal proportions, and, when often repeated, leads to structural changes in the organ.

Severe contusions over the region of the liver sometimes cause a hyperæmia, which may result in suppurative or in indurative inflammation.

In hot climates and in malarious districts, active and chronic hyperæmia of the liver are frequent, and often cause structural lesions. In scurvy, also, the liver is sometimes congested.

Cessation and suppression of the menses, and of hæmorrhoidal bleeding, may cause hyperæmia of the liver.

In all these varieties of active congestion, the liver is enlarged, of a deep red color, and blood flows freely from its cut surface.

The passive congestions of the liver are produced by some obstruction to the current of blood in the hepatic veins. Valvular diseases of the heart, emphysema, and fibrous induration of the lungs, large pleuritic effusions, intra-thoracic tumors, angular curvature of the spine, aortic aneurisms pressing on the vena cava, and constrictions of the vena cava and of the hepatic veins, may all produce a chronic hyperæmia of the liver. In all these cases, as the congestion affects principally the hepatic veins, we find the centre of each acinus congested and red, while its periphery is lighter colored. This gives to the liver a mottled or nutmeg appearance. The liver-cells in the centre of each acinus are frequently colored by little granules of red or black pigment, and the cells at the periphery become fatty, so that the nutmeg appearance is still more pronounced. A liver in this condition is usually of medium size, but may be smaller or larger than normal.

When the congestion is long continued, the veins at the centre of each acinus may become dilated, the hepatic cells in their meshes become atrophied, so that the centre of each acinus consists only of dilated capillaries and new connective tissue. The liver then becomes small, and has a finely granular appearance. But I have seen the liver enlarged while these changes existed.

In other cases, the obstruction of the hepatic veins does not produce the irregular nutmeg congestion. The entire organ is

congested, of a dark red color, of very firm consistence, somewhat increased in size; the intra-lobular veins and the central portion of the capillary plexus of each acinus are much dilated.

ATROPHY.

1. *Senile Atrophy*.—The liver may be diminished in size by any causes which interfere with its nutrition. Old age, insufficient food, new growths of the stomach, chronic poisoning with lead, mercury, arsenic, and antimony, are among the most common of such causes. The liver is found atrophied in all its dimensions, firm, the acini small.

2. *Yellow Atrophy*.—During pregnancy, after typhus fever, with phosphorus poisoning, and from unknown causes, we sometimes meet with severe symptoms, coma, delirium, jaundice, etc., during life. After death, the liver is found small, flabby, yellow, anæmic, its cells in a condition of fatty and granular degeneration. Our knowledge concerning the nature of the disease, its causes, and the relation between the symptoms and lesions, is very imperfect.

3. *Red Atrophy*.—In a few cases, symptoms of much the same character, but of longer duration, are accompanied by a different condition of the liver. The organ is very much atrophied in all its dimensions. It is of a bright or dark red color, mottled with rounded yellow spots. In some cases, the red and yellow colors are more irregularly distributed, so that a large part of a lobe may be yellow. In the red portions of the liver, the acini are atrophied; in the yellow portions, they are hypertrophied. In the yellow portions, the hepatic cells are large and fatty. The interstitial tissue and vessels are unchanged. The red portions consist of loose connective tissue, in which are small spaces filled with granular fat and bile pigment. There are also peculiar, long, branching follicles, lined with polygonal cells, of which the nature is obscure. Some authors regard red atrophy as a more advanced stage of yellow atrophy.

4. *Cyanotic Atrophy* may be produced by any cause which impedes the flow of blood from the hepatic veins. The liver is diminished in size, is very hard, of mottled red and yellow

nutmeg color, or of a uniform dark red. The veins at the centres of the acini are dilated; the liver-cells between them are atrophied, pigmented, and diminished in number. The peripheral cells are normal or fatty. There is a production of lymphoid cells at the centres of the acini.

Atrophy of the liver is also produced by obliteration or stenosis of the portal vein and its branches, by obstruction of the capillaries by pigment, and by obliteration of the capillaries.

Permanent obstruction of the hepatic or common bile duct may produce atrophy. The biliary ducts throughout the liver are dilated, the liver is stained at first yellow and afterward green, the consistence is soft, the size of the organ is much diminished, and the liver-cells are pigmented, fatty, and broken down.

Chronic perihepatitis, with great thickening of the entire capsule of the liver, sometimes produces marked atrophy with the symptoms of cirrhosis.

HYPERTROPHY.

Simple hypertrophy of the liver is to be diagnosticated with great caution. It is found with leucæmia, diabetes, phthisis, and cardiac disease, in malarious districts, and without known cause. The liver is increased in size, the acini are larger than normal, the liver-cells are increased in size or number, the blood-vessels are also large.

HÆMORRHAGE.

Extravasations of blood in the hepatic tissue, or, more frequently, beneath its capsule, are found in new-born children after tedious or forcible labors, and with atelectasis of the lungs. The blood may burst through the capsule, and be found in the peritoneal cavity.

In adults, such extravasations are rare. They are seen in the malignant, malarious fevers, especially in tropical countries; in scurvy and purpura; and in consequence of external violence. There are also a few curious cases recorded of such sudden hæmorrhages in which no cause was discovered. The extravasations of blood are beneath the capsule, or circumscribed

in the hepatic tissue, or in the form of a diffused hæmorrhagic softening. If the extravasation is small, it may be absorbed, leaving a cicatrix. According to Stokes, such hæmorrhages may produce gangrene of the liver.

INFLAMMATION.

The liver is often the seat of inflammatory changes. These changes affect the capsule of the organ, its interstitial connective tissue, or its cells; they involve either a portion or the whole of the liver.

PERIHEPATITIS.

The capsule of the liver and its prolongations along the vessels are frequently inflamed. The inflammation may be part of a general peritonitis, or may be confined to the liver.

When the inflammation is confined to the liver, it is dependent upon the different forms of hepatitis; upon new growths and hydatids of the liver; upon pleurisy of the right side; upon syphilis; rarely upon traumatic causes; and very frequently occurs without any assignable cause or previous symptoms.

Perihepatitis usually results in thickening of the capsule and the formation of membranous adhesions. These lesions are usually of little consequence, and frequently give rise to no symptoms during life. In some cases, however, the entire capsule is very much thickened, the liver is small and compressed, and the symptoms are those of cirrhosis.

In rare cases perihepatitis is purulent, and produces collections of pus shut in by false membranes.

The inflammation of the prolongations of the capsule on the vessels will be considered with diseases of the vessels.

CHRONIC PARENCHYMATOUS HEPATITIS.

Förster describes, under this name, a lesion which he says is usually confounded with cirrhosis.

The liver is at first large, congested, or anæmic. Following the stage of hypertrophy, a gradual, slow atrophy sets in. The organ is small, anæmic, yellow, and has a granular appearance. This granular appearance, however, is not like that of cirrhosis,

but is produced by the destruction of some of the acini, and the consequent undue prominence of others. The liver-cells are destroyed, or are small and granular.

This lesion may produce jaundice, ascites, and sometimes death.

INTERSTITIAL HEPATITIS.

Interstitial hepatitis is acute, and leads to the formation of abscesses; or chronic, and results in the formation of new connective tissue.

PURULENT HEPATITIS.

The study of the formation of abscesses in the liver has been very much confused by confounding two separate pathological conditions. (1) Abscesses formed by a purulent hepatitis; (2) abscesses formed by the irritation of emboli, and from thrombosis of the portal vein. The description of the first stages of the lesion is usually drawn from the second, and of the latter stages from the first of these conditions.

(1.) Idiopathic abscesses are of rare occurrence, except in tropical climates. In temperate climates, so few cases are seen that we know but little of their causation. A few cases are recorded in which severe contusions were the exciting causes of abscesses of the liver. Dysentery, with ulceration of the large intestine, is sometimes associated with single abscesses of the liver; but the exact relation between them is not well known. Inflammation and ulceration of the bile-ducts, excited by the presence of biliary calculi, or of intestinal worms, may produce multiple abscesses by the extension of the inflammation from the ducts to the liver tissue.

Of the earlier stages of idiopathic suppurative hepatitis but little is known. The abscesses are not seen in autopsies until after they are fully formed. They may then reach a large size, and are usually single, though sometimes multiple. They may be opened by the surgeon through the abdominal wall; or rupture spontaneously into the pleural cavity, the lungs, the abdominal cavity, the stomach and intestines, the hepatic vein, the

bile-ducts, the gall-bladder, the right kidney, the muscles and skin of the abdominal wall. Such abscesses usually prove fatal; but sometimes their artificial or spontaneous evacuation is followed by recovery.

(2.) Abscesses of the liver are formed in the same way as embolic abscesses in other viscera. There is thrombosis, from some cause, in the portal vein or its intestinal branches, or in the veins of the lungs, or in any of the veins belonging to the aortic system. A portion of such a thrombus finds its way either into one of the small branches of the portal vein, or of the hepatic artery. This sets up a suppurative inflammation of the adjoining portion of liver tissue. The thrombus simply acts as an irritant, and produces suppuration without infraction.

(3.) In thrombosis of the portal vein, the thrombi extend into the liver as far as the small interlobular veins. The veins are filled with a thrombus, which is either firm and white, or soft and puriform. Their walls are thickened, the inner surface becomes opaque and rough, and is sometimes lifted up by little collections of pus, the middle and external coats and the capsule of Glisson are all infiltrated with lymphoid cells. The liver-cells and the branches of the hepatic veins are compressed and degenerate from the pressure of the enlarged and inflamed branches of the portal vein, the liver-cells atrophy, break down, and disappear, and there is left an abscess filled with pus and debris.

CHRONIC INTERSTITIAL HEPATITIS.—CIRRHOSIS.

The interstitial connective tissue which accompanies the veins and bile-ducts, in their ramifications through the liver, is frequently the seat of a chronic inflammation which results in the formation of new connective tissue. It has been thought necessary by pathologists and clinicians to describe different stages of the disease which must succeed each other. Such a description is forced and without sufficient anatomical basis. We meet with different degrees and qualities of the lesion, but we do not know their relationship.

The appearances vary according to the amount of new con-

nective tissue, its distribution, its vascularity, and the condition of the hepatic cells.

In the least advanced condition, we find the liver of normal size or larger, not congested, firmer and smoother than usual, of a light red or yellow color, with a peculiar glistening reflex, like that of a waxy liver. The edges of the acini are surrounded by a light yellow streak, which is composed of connective tissue fibres, with a variable number of cells. The hepatic cells are usually normal, sometimes fatty.

In other cases, the liver is increased in size, its edges rounded, of a yellowish brown color, its surface slightly uneven, its capsule thickened. Groups of acini are surrounded by bands of reddish gray, vascular connective tissue.

When the lesion is far advanced, the liver is usually much diminished in size; sometimes, however, it is very much larger than the normal. The capsule is thickened, the surface is strikingly irregular, and thrown into nodules of variable size. These nodules are of a yellow color, or stained green by the bile. They are surrounded by a layer of dense white or pinkish connective tissue. The increase of connective tissue takes place about each acinus separately, or about groups of acini, or in both ways. The new tissue is composed of fibrous tissue alone, or is largely cellular. The degree of vascularity also varies, and gives different shades of color. The relative amount of cells and fibres in the new connective tissue does not seem to indicate an early or late stage of the disease; for in cases where the lesion is of long standing, and the liver atrophied, the cells are sometimes abundant. The hepatic cells usually become fatty, or may be waxy or pigmented, or are broken down and destroyed. The pigment is derived from the bile, and is of a yellow or green color; or from the blood, and is reddish brown.

The smaller branches of the portal vein are narrowed and compressed by the new connective tissue. Sometimes the main trunk and large branches are dilated. When the new connective tissue has replaced the hepatic structure, the capillary venous plexus is entirely destroyed, and there are only irregular vessels which can be injected from the portal vein and

hepatic artery. In some cases, the portal vein and its large branches have been found filled with large thrombi.

The hepatic artery becomes enlarged and its branches are more extensive.

The hepatic veins are usually unchanged, with the exception of their capillaries, which are gradually destroyed.

The bile-ducts are compressed by the new tissue; the mucous membranes of the large ducts may be inflamed and swollen, and the gall bladder is often thickened and adherent.

The obstruction of the portal circulation, produced by cirrhosis, gives rise to a number of secondary lesions.

There are a number of veins which can afford a collateral circulation between the portal vein and the vena cava. There are normally anastomoses between the internal and inferior hæmorrhoidal veins; between the left coronary and the œsophageal and diaphragmatic veins; between the hæmorrhoidal and vesical veins; between the coronary vein of the stomach, the gastro-epiploic veins and the renal veins; between the superior mesenteric and the left renal vein; between the vasa brevia and the left phrenic vein. All these vessels, however, are seldom enlarged in cirrhosis.

There are sometimes large anastomoses between the veins of Glisson's capsule and the diaphragmatic veins.

In rare cases, a very peculiar dilatation of the cutaneous veins about the umbilicus is observed. The enlarged veins form a circular network around the umbilicus, or a pyramidal tumor alongside of it, or all the veins of the abdominal wall, from the epigastrium to the inguinal region, are dilated. This condition is produced by the congenital non-closure and subsequent dilatation of the umbilical vein and its anastomoses with the internal mammary, epigastric and cutaneous veins. According to Sappey, it is not the umbilical vein which is dilated, but a vein which accompanies the ligamentum teres.

There is very frequently also a dilatation of the veins of the abdominal wall, which has a different cause. It is produced by the pressure of the fluid of ascites on the vena cava, and is found with ascites from any cause and with abdominal tumors.

Ascites is the most common secondary lesion of cirrhosis. It

begins at an early stage of the disease, and increases constantly. It usually precedes œdema of the feet, but both may appear at the same time. This fluid is of a clear yellow or brown, green or red; it is sometimes mixed with shreds of fibrine, and more rarely with blood. The peritoneum remains normal, or becomes opaque and thick, or there may be adhesions between the viscera.

The spleen is very frequently enlarged, and the enlargement may be very considerable. When it is not increased in size, this is usually due to previous atrophy of the organ, or to fibrous thickening of its capsule, or to hæmorrhages from the stomach and bowels, occurring just before death.

The stomach and intestines are often secondarily affected by the obstruction to the portal circulation.

Profuse hæmorrhage from the stomach and intestines may occur, and sometimes causes sudden death. The mucous membrane is then found pale, or congested, or with hæmorrhagic erosions. Sometimes the blood is infiltrated in the coats of the stomach and intestines. The mucous membrane of the stomach, and of the entire length of the intestines, is sometimes uniformly and intensely congested, and coated with mucus. In other cases both the mucous and muscular coats are pale, but very markedly thickened.

The causes of cirrhosis are not well known. It is a disease of adult life, and is often found in habitual drinkers; but there are many cases in which no cause can be discovered.

SIMPLE INDURATION OF THE LIVER.

This name has been given to a variety of chronic interstitial hepatitis, in which the new fibrous tissue has a different arrangement. It is produced in masses of considerable size, which replace the hepatic tissue, and are smooth and dense.

In a considerable number of cases, this lesion occurs in persons who have been the subject of constitutional syphilis.

The history of the disease is the same as that of cirrhosis.

SYPHILITIC HEPATITIS.

Diffuse chronic interstitial hepatitis occurs not infrequently in persons who have suffered from constitutional syphilis. Such a hepatitis may assume the ordinary characters of nodular cirrhosis of the liver, or it may assume a form which is peculiar and different from this. The difference consists merely in the distribution of the new fibrous tissue. Instead of surrounding single acini, or small groups, the new connective tissue surrounds larger portions of the organ, and thus gives it a lobulated instead of a nodulated form. There is at the same time perihepatitis, with thickening of the capsule; and from the thickened capsule dense fibrous septa extend into the liver. A lobe of the liver may be, in this way, divided up into two or more large subdivisions, or into a great number of small lobules. There are also, in some cases, gummy tumors formed in the fibrous septa. In other cases, the lobulation is less marked, but we find smooth, hard masses of fibrous tissue replacing portions of the hepatic tissue.

DEGENERATIONS OF THE LIVER.

THE FATTY LIVER.

In the human liver there is usually a certain amount of fat in the liver-cells, and this amount varies considerably under different conditions, without constituting a pathological lesion. The amount of fat in the liver-cells, and the extent to which it is distributed, may, however, reach such an extent as to be entirely abnormal.

The fat is found in the hepatic cells, in the form of globules of different sizes. A single cell may contain several small fat globules, or may be so distended with fat that only a narrow rim of cell-tissue is left. The accumulation of fat usually begins in the periphery of each acinus, next to the branches of the portal vein. The gross appearance of the organ varies a good deal in different cases. It is usually increased in size, flattened,

and its edges are rounded; but the increase may also be in thickness, and the edges may remain sharp. In other cases, the liver is of normal size, and in others again is atrophied. The color is red, mottled with large patches of yellow on the surface; in such cases, only the yellow portions of the liver are fatty. Or, the entire liver has a mottled red and yellow color, resembling the nutmeg-liver; this is produced by fatty degeneration of the cells at the periphery of each acinus, with some congestion of the veins at its centre. Or, the entire liver is of a pale yellow color, and contains very little blood. Or, there may be yellow or green staining with bile pigment.

The consistence of the liver also varies. It may be very soft and greasy, or it may be firm, or even hard. This firmness may depend on a different character of the fat, or on the coexistence of chronic interstitial hepatitis, or on waxy degeneration of some of the liver-cells.

Of the causes of fatty liver we are, in a great measure, ignorant. In persons affected with pulmonary phthisis, and in habitual drinkers, the lesion is so often met with that it may be considered as dependent upon those conditions; but it is also seen associated with many other diseases.

Fatty liver may produce jaundice and symptoms of bile-poisoning.

THE WAXY LIVER.

The liver is not infrequently the seat of an infiltration with a peculiar transparent, homogeneous, glistening substance. This substance, called amyloid, waxy, or lardaceous material, has the property of being stained of a deep-red color by a weak solution of iodine. It has been supposed to be analogous to cellulose; to be an albuminous substance; to be dealkalized fibrine, etc.; but as yet there is no exact knowledge of its real nature. In the liver, the waxy material affects, first, the walls of the branches of the hepatic artery; then the liver-cells midway between the centres and peripheries of the acini; then the centres of the acini; and lastly, the whole of the acini. The

walls of the venous capillaries may also be infiltrated. According to Wagner, only the hepatic artery and the venous capillaries become waxy, while the hepatic cells are only atrophied. Most observers, however, place the lesion in the hepatic cells.

The gross appearance of a waxy liver varies according to the extent of liver-substance invaded, the way in which the waxy material is distributed, and the coëxistence of fatty degeneration, and interstitial hepatitis.

There may be only a few waxy cells scattered here and there in different acini, and the rest of the liver tissue be normal. Then the liver will have its normal size and appearance. Or, if the degeneration affect the greater part of the liver, the organ will be much enlarged, of a peculiar reddish, translucent color, and of a firm, waxy consistence. Or, in rare cases, there are a few circumscribed nodules of waxy cells, while the rest of the organ is not waxy. Or, if there is at the same time fatty degeneration of the other hepatic cells, the consistence and color of the liver will be modified. Or, if there is interstitial hepatitis, the liver will be nodulated or lobulated. Waxy degeneration of the liver is frequently associated with the same degeneration of the spleen, kidneys, and intestines. It does not produce ascites, except in a few cases, which seem to depend rather on general hydremia than on obstruction to the portal circulation. It may in exceptional cases cause jaundice.

Waxy degeneration of the liver, as of other organs, is the result of chronic inflammation and suppuration, especially of the bones and lungs, and occurs in consequence of constitutional syphilis. It is also sometimes produced by long-continued malarial poisoning.

THE PIGMENT LIVER.

Malarial poisoning, whether it appears in the form of remittent or intermittent fever, produces in the blood a variable amount of pigment. This pigment, which is found most abundantly in the blood of the portal vein, is of black, brown, or reddish color. It is found in the form of small granules em-

bedded in transparent, finely granular cell-bodies of irregular shape, and in larger flakes of pigment alone. There are also colorless cells, like those holding pigment, which contain no pigment. The white globules of the blood are frequently also increased in number. These pigment bodies are found in the greatest number in the blood of the spleen and liver. These organs, therefore, assume a black or olive-brown color. The liver is usually of normal size, and the hepatic cells are normal; but it may be hypertrophied or atrophied, and the liver-cells may be fatty, or waxy, or cirrhotic. The pigment is found in the venous capillaries and in the branches of the hepatic artery.

This pigmentation is most constantly found in the spleen, but in exceptional cases is found in the liver and not in the spleen. In marked cases, the brain, lungs, intestines, kidneys, and subcutaneous connective tissue are found of a brownish color, and their capillaries contain pigment.

In the brain, the pigment masses may block up the small arteries and produce numerous punctate apoplexies. The black masses of sulphate of iron, produced by decomposition, can be distinguished by their blue color when treated with hydrochloric acid and ferro-cyanide of potash.

GRANULAR, OR PARENCHYMATOUS DEGENERATION.

We find a peculiar change in the liver-cells as an accompaniment of several different pathological processes. The hepatic cells are at first filled with fine granules, soluble in alkalies, and apparently of albuminous nature. At a later stage, the cells are filled with coarser and glistening granules, soluble in alcohol and æther, and of fatty nature.

These changes in the hepatic cells are found as a result of impeded circulation, of infectious diseases, and of poisons, or of general disturbances of nutrition.

Thrombosis of the portal vein, abscesses, and cirrhosis may produce granular degeneration of circumscribed portions of the hepatic tissue.

Pyæmia, septicæmia, erysipelas, epidemic cerebro-spinal

meningitis, acute miliary tuberculosis, acute articular rheumatism, typhus and typhoid fever, yellow-fever, cholera, the acute exanthemata, are all frequently accompanied by this change in the liver.

Poisoning by phosphorus, by arsenic, and by antimony produces this lesion in a marked degree. It will be found described more particularly under the head of poisons.

Chronic pulmonary diseases and anæmia, from any cause, may produce the same lesion in the liver.

THE HEPATIC ARTERY.

The hepatic artery, in rare cases, is the seat of aneurismal dilatations, which may attain a large size. Such aneurisms may displace the liver, produce intense pain, compress the bile-ducts so as to cause jaundice, and usually rupture into the abdomen or stomach. The diagnosis of this lesion is not often made during life.

Embolism is not very common. It produces infarctions and abscesses.

THE PORTAL VEIN.

Thrombosis of the portal vein is a lesion frequently met with. It is occasionally produced by inflammation of the vein, but more frequently the phlebitis is produced by the thrombus.

Thrombi of the portal vein may be produced by :

1. Weakening of the circulation from general debility.
2. Diseases of the liver which destroy or compress branches of the portal vein. Cirrhosis, chronic atrophy, cancer of the liver, and dilatation of the bile-ducts are the most frequent causes. The thrombus usually occupies both the branches and trunk of the portal vein. It may fill the vessel either partly or completely. It adheres firmly to the wall of the vein, is firm and hard, or may be organized, and is of a grayish, blackish, or jaundiced color. Fresh red coagula may be attached as prolongations to the firmer clots. The vein is usually dilated, its walls are thickened, and may be calcareous.

In cancer of the liver, the thrombus may be produced by the pressure on the vein, or the new growth may project into the vein, and form part of the thrombus. In very rare cases, the portal vein and its branches have been found filled with a soft material composed of nucleated cells, while there was no new growth in the liver or other viscera.

3. Tumors, or new connective tissue, which compress the portal vein below the liver.

Chronic peritonitis, tumors of the stomach, pancreas, and omentum, and tumors or enlargements of the adjoining lymphatic glands, are the most frequent causes.

4. There may be no cause for the thrombus. This is especially the case when isolated branches of the portal vein become filled and obliterated by a thrombus, and in consequence of this, the portions of the liver supplied by these venous branches become atrophied, and the liver assumes a peculiar lobulated shape.

In all the conditions mentioned above, the tendency of the lesion is to the permanent occlusion of the branches of the portal vein which are filled with the thrombus.

This obliterative form of thrombosis is usually attended by ascites, enlargement of the spleen, enlargement of the abdominal veins, rarely by jaundice, sometimes by hæmorrhage from the stomach and bowels.

Thrombi in the portal vein, instead of becoming hard and closing the vessels, may become soft and broken down; while the wall of the vein becomes the seat of a suppurative inflammation. The thrombi are then of a reddish, grayish, or yellow color; a thin layer adheres to the wall of the vein; while the rest of the thrombus is partly solid, or converted into a puriform fluid. Such thrombi, when they fill up the smaller veins, produce granular degeneration of the hepatic cells and abscesses.

The causes of this form of thrombosis are various.

(1.) Traumatic. Medical literature has so far only contained the single case, reported by Lambton, of thrombosis produced by a fish-bone. The bone was three centimetres long, was impacted in the head of the pancreas, penetrated the anterior wall of the superior mesenteric vein obliquely from above down-

ward, and from before backward, traversed its interior, and was lodged in the posterior thickened wall of the vessel. The mesenteric vein at this point was filled with a grayish thrombus, which was continued into the portal vein and its hepatic branches, and was partly firm and partly broken down. There was a spot on the posterior wall of the stomach, near the pylorus, which was considered to be the point of exit of the fish-bone.

To this case I am able to add a second observed in Bellevue Hospital in 1867, reported by Dr. Janeway, and examined also by myself. A man, aged forty-seven, about four weeks before his death was exposed to cold and wet. He was attacked with pain in the right side and cough, with the physical signs of pleurisy on the right side. A diarrhœa set in; the man sank into a typhoid condition; the abdomen became tympanitic; there was tenderness over the right iliac fossa; the urine contained albumen and granular casts.

Autopsy.—The brain and heart were normal. The left lung was bound down by old adhesions; in the right pleural cavity was a small amount of serum.

The abdomen contained purulent serum, and the viscera were coated with recent fibrinous exudation. The stomach and intestines were normal.

The spleen weighed nine ounces. The kidneys weighed twelve ounces, and were the seat of advanced chronic diffuse nephritis.

The liver weighed six and a half pounds. There were numerous small abscesses in the right lobe; two abscesses in the left lobe. The left division of the portal vein was filled with a firm red and white clot, over an inch long; the right division of the vein was lined with a firm thrombus; its walls were thickened. In this part of the vein, and in its hepatic branches, was a good deal of puriform fluid.

There was a fish-bone, two inches long, half in the mesenteric and half in the portal vein. The centre of this bone was covered by a thrombus.

The specimen is preserved in the museum of Bellevue Hospital.

In young infants, the ligation of the umbilical vein, instead

of producing a firm thrombus and obliterating the vessel, sometimes produces a soft, puriform thrombus which extends into the portal vein. The walls of the vein are thick and succulent; there are frequently abscesses in the liver and general peritonitis.

2. Ulceration of the intestines and stomach sometimes produces thrombosis of the veins leading from the part inflamed, and the thrombi are continued into the portal vein, or fragments of them are carried into it.

3. Abscesses of the spleen sometimes produce thrombosis of the splenic vein, which is continued into the portal vein.

4. Suppurative inflammation of the mesentery, and of the mesenteric glands, may produce thrombosis of the mesenteric and portal veins.

5. Inflammation and ulceration of the bile-ducts caused by gall-stones occasionally produce thrombosis of the branches of the portal vein.

6. In many cases of thrombosis and inflammation of the portal vein no cause can be discovered for the lesion.

In all the varieties of soft thrombi and phlebitis of the portal vein, we usually find jaundice, sometimes abscesses of the liver, sometimes enlargement of the spleen, and, as a rule, acute symptoms during life.

RUPTURE OF THE PORTAL VEIN.

This is a very rare lesion, of which only a few cases are recorded. In some of the cases there was fatty degeneration of the wall of the vein.

DILATATION OF THE PORTAL VEIN.

Dilatation may affect the trunk, branches, or roots of the portal vein, but very rarely the entire vessel.

It is caused by destruction of the capillaries of the liver from chronic atrophy and cirrhosis; and from occlusion of the vein by thrombi, by cancer, and by concretions.

The dilatation is generally uniform, sometimes varicose.

CALCIFICATION OF THE PORTAL VEIN.

Chronic endophlebitis may occur with the production of atheromatous degeneration and calcification of the portal vein and its branches. It affects separate patches of the wall, or the entire wall for a considerable distance.

This lesion of the vein may produce thrombosis.

THE HEPATIC VEINS.

The hepatic veins are found to present the same lesions as the portal veins, but much less frequently.

Dilatation of these veins is produced by any obstruction to the passage of the venous blood into the heart.

Chronic phlebitis and hard thrombi are produced by perihepatitis. The wall of the vein is thickened; gelatinous deposits or valvular projections on the inner surface of the vein may narrow or obliterate it. There may be apoplexies in the tissue of the liver.

Soft thrombi and suppurative inflammation of the branches of the hepatic vein are sometimes produced by abscesses in the liver.

NEW GROWTHS OF THE LIVER.

Fibroma.—A single case of a fibrous tumor is described by Luschka, as occurring in the liver of a young child.

Chondroma in the liver is of rare occurrence. It is secondary to chondroma in other regions of the body.

Angioma.—Small cavernous tumors are of frequent occurrence in the liver. They form irregularly-shaped, sharply-defined, reddish black spots in the liver-tissue, from the size of an acinus up to that of a man's fist. They are composed of a mesh-work, formed of connective tissue and smooth muscle; the cavities of this mesh-work are lined with epithelium, resembling that of the veins, contain venous blood, and can be injected from the portal vein and the hepatic artery. According to Virchow, the tumor is formed by the dilatation of all the veins of one or more liver acini. Rindfleisch, on the other hand, holds

that the growth is formed by an interlobular hyperplasia of connective tissue, followed by a cavernous metamorphosis.

Leukæmic New Growths are found in the liver in two forms. The organ is increased in size, sometimes very largely. The liver-cells are increased in size and number, so that the acini are larger. (1.) The lymphatic new growth takes the form of an infiltration, which accompanies the portal vein and its branches into the acini. The new growth then forms branched figures of a grayish white color, and of considerable size, or of a fine gray or white tracery, corresponding to different acini.

(2.) In other cases, the new growth takes the form of small, rounded, miliary tumors, of a grayish white color. They resemble miliary tubercles, but are very soft, almost fluid.

Not unfrequently these two forms are combined in the same liver. The new growth in both cases has the structure of lymphatic glandular tissue.

Adenoma.—Under this name authors describe two distinct varieties of tumor. The first consists in a new growth of liver tissue, the second in a new growth of follicular glandular tissue.

1. The first form occurs as one large or several small tumors, scattered through the liver. The tumors are inclosed in a fibrous capsule, or are continuous with the liver tissue. They are of minute size, or may measure 3 or 4 inches in diameter. They are of a grayish brown color, and of firmer consistence than the surrounding liver tissue. They are composed of cells, resembling the hepatic cells, but sometimes larger, and sometimes containing several nuclei. These cells are arranged in acini, like those of the liver, and the vessels and sometimes the bile-ducts of the liver ramify in the larger tumors.

2. The second form of adenoma is composed of follicles or tubules, filled with small polygonal epithelium. The tumors are usually multiple, small or large, inclosed in a fibrous capsule. They may undergo fatty degeneration, or may suppurate. It is a question whether they are not really epithelial cancers.

Tubercle.—In general tuberculosis, the liver is frequently the seat of numerous small, miliary, grayish, transparent granulations, which may be too small to be seen with the naked eye. These are found in the capsule and accompanying the vessels.

Occasionally, in children, such tubercular tumors become larger and cheesy; it is then difficult to distinguish them from syphilitic tumors.

Syphilitic Tumors are found in connection with interstitial hepatitis or alone. If interstitial hepatitis is present, the gummy tumors are found embedded in fibrous bands. They are small, or a number may be aggregated together, so as to form a tumor as large as a man's fist. They are of a grayish color, and are composed of round and fusiform connective tissue-cells. They usually, however, very soon undergo cheesy and fibrous degeneration, and are then of an opaque white or yellow color; the cellular elements disappear, and we find nothing but a fibrous or cheesy mass. In other cases, the tumors soften and break down, so that they may resemble abscesses.

Less frequently, there is no interstitial hepatitis, and we find a number of small, yellow, cheesy tumors, scattered through the liver. I have seen, in one case, large, white, gummy tumors without interstitial hepatitis, which, to the naked eye, resembled cancerous nodules.

In infants, a very minute form of these tumors is found. They appear as numerous, very small, white dots on the surface of the organ.

+ *Cysts* are usually produced by a dilatation of the bile-ducts. They are small, multiple, contain serum, mucus, and epithelium. New-formed cysts in the connective tissue are also described.

Sarcoma of the liver is not common. It is almost always secondary. The most frequent forms are melanotic or spindle-celled sarcomata.

Carcinoma.—The liver is the most frequent seat of secondary cancerous growths. Primary carcinoma of the liver is very rare. The primary tumor is often small, while the secondary growth in the liver is very large, so that the former may be overlooked. In some cases, the primary tumor is situated in the wall of the gall-bladder.

All the varieties of cancer, simple, medullary, scirrhus, melanotic, colloid, flat and cylindrical epithelial, are found in the liver; but simple cancer, either hard or soft, is the most frequent. It occurs in the form of nodules, or of a uniform infil-

tration of the liver tissue. The nodules are white or yellow, from fatty or cheesy degeneration, or red, from hæmorrhages. They are single or multiple, large or small; they may project from the surface of the liver in the form of nodules, or be even with it, or depressed. They are often the seat of hæmorrhages, which may burst through the peritoneum into the abdominal cavity. They are hard or soft, according to the relative proportion of fibrous tissue and cells. The cancerous nodules may undergo fatty or cheesy degeneration; they may become inflamed and purulent; they may soften at the centre, so as to form cyst-like cavities filled with viscid or thin serum. When the new growth infiltrates the organ, it is usually firm, white, or pinkish, and may occupy a large portion of the liver. The liver itself may be of medium size, or may attain immense dimensions. The hepatic cells may remain unchanged, or may become fatty, or be stained with bile.

The portal vein and its branches may become filled with thrombi, and with portions of the new growth. Either the growth in the liver erodes and projects into the vein, or the wall of the vein is unchanged, while its cavity is filled with the new growth; or, in rare cases, the veins alone contain the new growth. The hepatic veins and their branches usually remain exempt from this lesion.

The lymphatic ducts on the surface of the liver may be distended with cancerous growth, and the neighboring lymphatic glands are frequently enlarged and infiltrated.

The bile-ducts may remain unchanged, or become compressed and obliterated, or be dilated and rupture, or nodules of new growth may form in their walls.

Alveolar or colloid cancer of the liver is rare. It usually extends to the organ from some of the abdominal viscera. It has the appearance of a gelatinous, semi-translucent mass, replacing larger or smaller portions of the organ. Its minute structure is a fibrous stroma, forming alveoli filled with gelatinous matter, and a variable number of cells.

Lymphangioma Cavernosum.—Two cases of secondary tumors of this character have been observed in the liver. The

growths were composed of anastomosing canals, lined with epithelium, and containing peculiar hyaline bodies. The exact nature of these growths in the liver, as well as elsewhere, is still undetermined.

PARASITES.

Echinococcus.—This form of parasite occurs frequently, forming the so-called hydatids of the liver.

It is one of the stages of development of a small tape-worm, the *Tænia echinococcus*, which inhabits the intestines of the dog.

We find in the liver cavities lined with a layer of gelatinous material, to the inner wall of which are attached the heads of the echinococcus; or, the cavities are filled with a multitude of little cysts, from the inner surface of which grow the heads. Or, the cysts may be sterile, and we find the gelatinous material without any of the heads. Or, the contents of the cyst degenerate, and we find their contents mixed with fat, or pus, or bile, or blood, or their walls may be thickened with fibrous tissue, or be calcified.

In New-York, the lesion is a rare one, and we usually find only one or two cysts with degenerated contents.

In other countries, the lesion is much more common and formidable. The cysts reach an enormous size, the veins of the liver may be compressed and filled with thrombi, the bile-ducts compressed and ulcerated. So much of the liver tissue may be replaced by the hydatids, that the patient may die from this cause alone. Very frequently there is local peritonitis, and adhesions are formed between the liver and the surrounding parts.

In some cases the cysts rupture, and their contents are emptied into the peritoneal cavity, the stomach, the intestines, the pleural cavity, or the lung tissue. Sometimes the cysts perforate the bile-ducts, the vena cava, or some of the branches of the portal or hepatic veins. Sometimes the abdominal wall is perforated and a fistula formed between the cavity in the liver and the skin.

A rare and peculiar development of the parasite was first described by Virchow, under the name of *Echinococcus multilocu-*

laris. The cysts are found in large numbers, and arranged, not within each other, but side by side, so that the mass has the appearance of an alveolar, gelatinous tumor. This mass is very hard. It is composed of dense fibrous tissue, forming small anastomosing canals filled with gelatinous material. In this gelatinous material the heads or hooklets of the echinococcus may be present or absent. On the surface of the liver the lymphatics are sometimes enlarged and filled with the same material.

Distoma hepaticum.—This parasite has a flat, oval, leaf-shaped body, 3 cent. long, 12 mm. wide, and is of a dirty brown color. It is very seldom seen, and has no clinical history.

Distoma lanceolatum.—This worm has a thin body, about 9 mm. long. It is found in the gall-bladder and bile-ducts. It is of very rare occurrence.

Pentastoma denticulatum is the undeveloped form of the pentastoma tænioides, a parasite which inhabits the nasal cavity of dogs and of some other animals. In the liver of man and of the herbivorous animals, it is found in the form of small, rounded calcified cysts. The cysts contain fat, calcareous matter, and the remains of the dead parasite.

The Ascaris lumbricoides sometimes finds its way from the intestines into the bile-ducts. It may cause complete occlusion of the biliary passages, or dilatation, or ulceration, or the formation of abscesses in the liver.

THE BILIARY PASSAGES.

Catarrhal Inflammation most frequently attacks the lower portion of the common duct and the gall-bladder. In the acute form, it leaves but few changes after death. An abnormal coating of mucus is almost the only post-mortem lesion. If the inflammation, however, becomes chronic, the wall of the bile-duct is thickened and its cavity obstructed. In consequence of this, dilatation and ulceration of the bile-ducts may ensue.

The gall-bladder may be inflamed by itself or in connection with the biliary passages. If the disease is chronic, the wall of the gall-bladder is thickened, its duct may be closed, dilatation, ulceration, the production of gall-stones, calcification, and atrophy may ensue.

Inflammation of the stomach and duodenum, hyperæmia, and inflammation of the liver, concretions, and parasites, are the usual causes of catarrhal inflammation of the biliary passages.

Suppurative and Croupous Inflammation may attack the biliary passages and produce infiltration of pus in their walls, and purulent fluid in their cavities; or flakes and tubular casts of fibrine on their walls; or infiltration of their walls with fibrine, and diphtheritic ulceration.

These lesions are most frequently observed in typhoid and typhus fever, pyæmia, cholera, and with obstructions in the bile-ducts. They also occur under unknown conditions.

These forms of inflammation may produce perforations of the ducts or bladder, with escape of bile, and peritonitis; or fistulous openings between the gall-bladder and the duodenum, colon, and stomach, or through the abdominal wall. Or the inflammation may extend to the liver tissue and produce abscesses. Or the portal vein may be inflamed, and even perforations be formed between it and the bile-ducts. Constrictions of the biliary passages may also be produced by the same causes.

Constriction and Occlusion are produced by inflammation of the ducts themselves, by new growths in their walls, by concretions or parasites in their cavities, by changes in the hepatic tissue, by aneurisms.

The obliteration of the smaller bile-ducts produces no marked lesions. When the ductus communis, or the hepatic duct, is obstructed, the ducts throughout the liver are frequently dilated and the liver tissue bile-stained. When the cystic duct is obstructed, the gall-bladder is dilated.

Dilatation of the bile-ducts is usually produced by strictures in the ways just mentioned, or by calculi. When calculi have produced the dilatation, this condition may sometimes continue after they have found their way into the intestines. Sometimes, however, we meet with very marked dilatation of the bile-ducts without being able to make out any present or past obstruction. The dilatation may affect only the common and hepatic ducts, or it may extend to the smaller ducts in the liver, which are then dilated uniformly or sacculated. They

may contain bile, mucus, or calculi. The liver is at first enlarged, but may afterward atrophy.

The gall-bladder may be dilated in consequence of obstruction of the common or the cystic ducts. In the latter case, it may reach an immense size, and form a large tumor in the abdominal cavity. The dilatation is generally uniform, the bladder retaining its normal form; sometimes, however, there are diverticula, which are usually produced by calculi.

If the obstruction to the hepatic duct is incomplete or movable, the gall-bladder contains bile, and often calculi. If the obstruction is complete, the contained fluid gradually loses its biliary character, and becomes a serous or mucous fluid of a light yellow color.

The walls of the bladder may be of normal thickness, or thinned, or thickened, or calcified.

If the obstruction is due to a calculus, this may pass into the intestine and the gall-bladder be suddenly emptied. Usually, the bladder fills again, owing to its loss of contractile power.

NEW GROWTHS.

Fibroma has been observed in rare cases in the gall-bladder and in the common duct.

Carcinoma occurs both as a secondary and a primary growth. It is more frequent in the gall-bladder than in the bile-ducts. Simple cancer, villous, cylindrical, epithelial, and colloid cancer have all been observed in this situation.

BILIARY CALCULI.

These bodies are of common occurrence. They are found usually in the bladder; sometimes in the hepatic, cystic, and common ducts; least frequently in the small ducts in the liver. In the gall-bladder from one to 7800 calculi have been counted.

They are composed:

1. Principally of cholesterine. They are round, oval, or faceted, single or multiple, of a pure white color, or tinged in various shades by bile-pigment.

2. Of cholesterine, bile-pigment, and sometimes carbonate and phosphate of lime. They are usually dark-colored, brown, red, black, or green, round or faceted.

3. Principally of bile-pigment. These are small, not numerous, dark-colored.

4. Of carbonate of lime. These are uncommon, have a nodular surface, and a clear, crystalline fracture.

Most calculi consist of a nucleus and body. The nucleus is composed of cholesterine and bile-pigment; or, more rarely, of some foreign body. A dead parasite, a needle, and stones of fruit have been found as nuclei. The body of the gall-stone is homogeneous or laminated.

Biliary calculi in the gall-bladder usually produce no symptoms, and are only discovered after the death of the patient. In the hepatic and common ducts they may obstruct the flow of bile and produce fatal jaundice, or they pass from time to time into the intestine, and produce biliary colic. If they are impacted in the cystic duct, they may produce dilatation of the gall-bladder.

THE SPLEEN.

MALFORMATIONS.

The spleen may be entirely absent in acephalous monsters, and with defective development of the other abdominal viscera. Very rarely it is absent in persons who are otherwise perfectly developed.

Small accessory spleens, not larger than a walnut, and situated close to the spleen, are not unfrequent.

The existence of two spleens of equal size has been observed in one case.

The spleen may be on the right side when there is transposition of the other viscera.

HYPERTROPHY, ATROPHY.

In cases of typhoid fever, pyæmia, cholera, the exanthemata, malarial fevers, etc., the spleen is usually enlarged, soft, and of

a dark-red color. The exact nature of this enlargement is not known. If the patient recovers, the organ returns to its normal size.

A more chronic and persistent enlargement of the spleen is found with intermittent fever, heart-disease, leucæmia, with chronic diseases, and without known cause. The spleen is then simply increased in size, without other change from its normal appearance; or the fibrous stroma is disproportionately increased; or there is an unusual hyperæmia.

Atrophy of the spleen occurs as a senile change. We also frequently find it at the autopsy, without any cause to account for it.

CHANGES IN POSITION.

The spleen may be pressed downward by any increase in the contents of the thorax. It may be fastened by adhesions to the concave surface of the diaphragm, so that its long axis is nearly horizontal, instead of vertical. It may be displaced by changes in the contents of the abdominal cavity. If the organ is increased in size, it frequently becomes tilted, so that its lower border reaches the right iliac region. If the ligaments of the spleen are too long congenitally, or if they are lengthened by traction, and if the organ is at the same time increased in weight, it may become very movable. It sinks downward, with its hilus turned upward; or it may be rotated on its axis. It may, after a time, cause atrophy; or it may compress the duodenum.

RUPTURE.

The spleen may be ruptured by severe blows or falls. Fatal hæmorrhage usually ensues, but sometimes the patient recovers.

Rupture has also been observed in a few cases of typhoid fever, remittent and relapsing fever, and of cholera.

INFLAMMATION.

It is a very frequent occurrence to find the capsule of the spleen thickened and adherent to the neighboring viscera. For the most part, the cause of these changes cannot be ascertained.

Inflammation of the spleen itself occurs in the form of abscesses or infarctions. The abscesses may be traumatic, or produced by infarctions, or occur without known cause. The abscesses are single or multiple. They may reach a great size; they may rupture into the abdominal cavity, the pleura, the lungs, the stomach, the colon, or the kidneys.

Infarctions of the spleen are very common. Vegetations on the heart-valves, and thrombi in some of the veins, are the usual causes. Sometimes no source for the embolus can be discovered. We find one or more wedge-shaped portions of the spleen, at its surface, at first infiltrated with blood, and afterward converted into a yellow, cheesy mass, or into a fibrous cicatrix. Less frequently suppurative inflammation ensues, and an abscess is formed. In other cases, again, the infarction becomes gangrenous, and rarely the gangrene may involve the greater part of the spleen. In consequence of the gangrene, fatal peritonitis may ensue.

DEGENERATION.

Waxy degeneration, or rather infiltration of the spleen, is very common. It affects the entire organ, or only the Malpighian bodies. In the first case, the organ is large, firm, smooth, reddish, and waxy-looking. Nearly the entire tissue is infiltrated with the waxy substance, and reacts with iodine. If only the Malpighian bodies are affected, the spleen appears to be studded with small, round, grayish bodies looking like grains of boiled sago. We then find the vessels and cells of the Malpighian bodies infiltrated with the waxy matter. This form is called the sago spleen.

NEW GROWTHS.

Fibroma.—Small, rounded nodules of fibrous tissue have been seen in the spleen in a few cases.

Cysts are rare. They are single or multiple, and contain serum. Single dermoid cysts, filled with fat and hair, have also been seen.

Tubercles are found in the capsule and in the splenic tissue in the form of gray granulations.

Syphilitic Gummata have been seen in the spleen.

Lymphatic Tumors in the form of white nodules are found in leucæmia and in pseudo-leucæmia.

Pigment.—In persons who are the subjects of malarial poisoning, the spleen is usually enlarged, and of a black color. The position and source of the pigment, which causes this color, is still uncertain.

Carcinoma is very rarely primary in the spleen. More frequently it is metastatic; sometimes directly infiltrated from new growths in the stomach.

PARASITES.

Echinococci are sometimes found in the spleen, either as isolated bodies or as composite cysts.

Pentastomum denticulatum in a calcified condition has been seen by Wagner.

THE PANCREAS.

MALFORMATIONS.

The pancreas may be entirely absent in anencephalous and double monsters, and in congenital umbilical herniæ. The pancreatic duct may be double; it may open into the duodenum at some distance from the biliary duct, or into the stomach. The head of the pancreas may be unduly developed, and sometimes even completely separated from the rest of the organ, opening into the duodenum with a duct of its own.

CHANGES IN FORM AND POSITION.

The pancreas is so firmly bound down that its position is not often changed. Sometimes, however, it is found pressed downward by tight lacing, displaced by aneurisms, or contained in umbilical and diaphragmatic herniæ.

Atrophy of the pancreas is produced by the pressure of

tumors, aneurisms, etc. ; by obstruction of its duct ; and is also found as an idiopathic process. The atrophy may reach such a degree that the gland can no longer be distinguished with the naked eye. This extreme degree of atrophy is sometimes found in diabetes. A moderate degree of atrophy seems to be a regular senile change.

INFLAMMATION.

Suppurative Inflammation of the pancreas is very rare. The organ is found infiltrated with pus, or contains abscesses, or is surrounded by pus. The collections of pus thus formed may spread into the neighboring soft parts, and perforate into the abdominal cavity, the stomach, or the intestines.

Some of the cases described as abscesses seem to be cysts with purulent contents. The recorded cases of suppurative inflammation of the pancreas are so few that hardly any thing is known of its cause.

Chronic Interstitial Inflammation of the pancreas results in the production of new fibrous tissue, with induration and subsequent atrophy of the organ. The contraction of the new fibrous tissue may obliterate the pancreatic ducts, and may also compress the ductus communis choledochus, so as to produce icterus and even dilatation of the bile-ducts.

DEGENERATION.

Waxy Degeneration affects, in rare cases, the blood-vessels of the pancreas ; the gland-cells are not involved.

NEW GROWTHS.

Carcinoma of the pancreas is not common. It occurs both as a primary and secondary growth.

As a primary growth, it usually takes the form of hard, fibrous nodules scattered through the glands, or of a uniform, scirrhous enlargement ; more rarely, colloid cancer and cylindrical-celled epithelial cancer have been observed. The new growth is con-

finer to the pancreas, or may spread to the duodenum, the stomach, or the neighboring lymphatic glands.

As a secondary growth, carcinoma usually invades the pancreas by continuous infection from the duodenum, stomach, or lymphatic glands.

THE PANCREATIC DUCTS.

Concretions of carbonate and phosphate of lime are frequently found in the pancreatic ducts. They are usually multiple, small, whitish, of rough and irregular shape. Sometimes, however, they reach a diameter of more than an inch. Besides these free concretions, the walls of the ducts are sometimes incrustated with salts of lime.

Such concretions may produce dilatation of the pancreatic ducts and large cysts, or, more rarely, abscesses.

Foreign Bodies.—Gall-stones sometimes find their way into the pancreatic duct. Ascarides have been found in the ducts in a considerable number of cases.

Dilatation of the pancreatic ducts, and the formation of cysts, take place in several different ways.

(1.) The entire duct may undergo a uniform, cylindrical dilatation. With this cylindrical dilatation we sometimes find associated small sacculi.

(2.) There may be sacculated dilatations at some point in the ducts. These dilatations form cysts of large size, as large even as a child's head. Their walls frequently become atheromatous and calcified. These cysts often become filled with blood, and may then be mistaken for aneurisms.

(3.) The small branches of the pancreatic duct may be dilated so as to form a number of small cysts. These cysts are filled with serum, mucus, pus, or a thick cheesy material.

THE SUPRARENAL CAPSULES.

MALFORMATIONS.

In anencephalous and other monsters the capsules may be entirely absent or much atrophied. Sometimes, in well-formed adults, the organs cannot be discovered.

There may be little rounded nodules loosely attached to the surface of the capsules and with the same structure as these.

If one of the kidneys is absent, or in an abnormal position, its capsule usually retains its proper position.

HÆMORRHAGE.

In children, soon after birth, it is not very infrequent to find large hæmorrhages in one of the capsules, converting it into a cyst filled with blood. The same lesion has been observed in a few cases in adults.

THROMBOSIS.

Klebs describes a case of capillary thrombosis of the cortex in both capsules in a woman after excision of the knee-joint.

INFLAMMATION.

The most frequent lesion of the capsules is their transformation into fibrous tissue and cheesy matter. The capsules are then large, their surface smooth or lobulated, or they are contracted and irregular. The normal structure of the gland is lost, and we find instead a mass of fibrous tissue in which are embedded cheesy nodules. The cheesy nodules may soften and become fluid, or may calcify. Either one or both capsules may be involved. The early stages of this lesion are obscure, and it is difficult to know how far chronic inflammation, and how far tuberculosis is the efficient cause.

Sometimes the medullary portion is replaced by a homogeneous, grayish, structureless substance, which later becomes cheesy.

Sometimes there are grayish nodules in the medullary substance, which become cheesy and may fuse together.

Sometimes there is a true suppurative inflammation with the formation of abscesses.

Our knowledge of all these conditions is very imperfect.

DEGENERATION.

Fatty Degeneration of the cortical portion of the capsules is the rule in the adult. In children under five years of age, it is a pathological condition.

Amyloid Degeneration may involve both the cortical and medullary portions. In the cortex, it usually involves only the walls of the blood-vessels; in the medulla, both the blood-vessels and the cells of the parenchyma may undergo this degeneration. The capsules are usually firm and of a grayish, semi-translucent color.

NEW GROWTHS.

Carcinoma of the suprarenal capsules is not common. It may be primary, but is much more frequently secondary. Either one or both of the capsules may be the seat of the new growth.

Sarcoma occurs as a primary and secondary growth. Probably many of the older cases described as cancers were really sarcomata.

Miliary Tubercles occur but rarely.

Cylindroma.—Klebs describes a growth of this character in one of the capsules, secondary to a tumor of the same kind in the supraorbital region. He gives to such tumors the name of lymphangioma cavernosum. The exact character of these growths is still obscure. They consist of irregular follicles and cavities, lined with epithelium, and containing peculiar hyaline, structureless bodies.

Cysts are found both single and multiple. They are usually situated in the cortex.

THE URINARY APPARATUS.

THE KIDNEYS.

MALFORMATIONS.

Entire absence of both kidneys is seen with high degrees of malformation of the entire body. Such *foetuses* are not viable. Absence of one kidney is found occasionally in adults, and seems to have no effect on their general health. The left kidney is more frequently absent than the right. The ureter belonging to the absent kidney is usually entirely wanting; but a small portion of it may be found attached to the bladder. The single kidney which is present is hypertrophied. Its ureter usually opens into the bladder in its proper situation, although Förster describes a case in which the kidney was on the right side, while its ureter entered the bladder on the left side. I have seen a case in which the single kidney was situated over the promontory of the sacrum. Its artery was given off from the aorta at the bifurcation of the iliac arteries, and its vein from the vena cava, near the bifurcation. Its ureter was large and short; it entered the bladder midway between the usual position of the two ureters.

It is not uncommon to find one kidney so much atrophied that hardly any remains of kidney tissue are to be found; nothing but fibrous tissue and fat. In these cases, however, some traces of a ureter will be found.

The *foetal* lobulated condition of the kidney often persists in adult life. Sometimes one or more of these lobules become excessively developed, and somewhat separated from the rest of the kidney, so that the organ may appear double. A single

case is recorded by Blasius in which there were two left kidneys, each with its vessels and ureter.

The kidneys are sometimes joined together. This is effected by a bridge of kidney tissue, uniting the lower ends of the two kidneys. Sometimes such a "horse-shoe" kidney is situated lower than usual over the lowest lumbar vertebra, or in the pelvis. The renal vessels are then given off from some abnormal point.

In other cases the two kidneys are united throughout, so as to look like a single misshapen kidney, with two or more pelvises. The kidneys thus closely united may be both situated on one side of the vertebral column, or in the pelvis. The arrangement and origin of their vessels vary according to the situation of the kidneys.

CHANGES IN POSITION.

The kidneys may have an abnormal situation, in which they are either fixed or movable.

The change in position is either lateral or downward. The most frequent situation is the promontory of the sacrum; but the kidney has been seen at the very bottom of the pelvic cavity. The vessels have an irregular origin and distribution corresponding to the changed position of the kidney. The kidney is firmly attached in its unusual position.

Movable or wandering kidneys are not congenital, but are found in adult life as a result of tight-lacing, pregnancy, over-exertion, and from unknown causes. They are more frequent in females than in males. The right kidney is the one more frequently movable; but both organs may be found in this condition. Sometimes a kidney, which is congenitally misplaced, is also movable.

The ordinary movable kidneys have their vessels regularly given off, but lengthened. The kidney tissue may be normal or may undergo any of the ordinary pathological changes.

Cases have been observed in which the kidney, by its unusual position, had compressed the ascending colon, and had caused thrombosis of the vena cava.

HÆMORRHAGE.

Hæmorrhages are produced in the kidney by wounds, and by severe contusions and falls, causing rupture of the kidney tissue. The blood collects in the kidney, beneath its capsule, or in the surrounding connective tissue.

Rokitansky describes a form of hæmorrhage produced by excessive congestion. The kidney tissue is torn by the diffused blood.

Smaller hæmorrhages are found with infarctions, purpura, and nephritis.

CHRONIC CONGESTION OF THE KIDNEY.

Any disease of the heart or lungs, or any mechanical cause in the thorax or abdomen, which obstructs the passage of venous blood into the right auricle, and of the arterial blood from the left ventricle, is liable to produce a condition of chronic congestion of the kidneys. Valvular diseases of the heart are the most common of these causes. The pregnant uterus, by its mechanical pressure on the veins, produces the same effect.

The clinical symptoms are a decrease in the quantity of the urine, with an increase of its specific gravity, together with a variable amount of albumen, hyaline casts, and sometimes blood in the urine.

The appearance of the kidneys is very characteristic. They are of normal size, or a little larger; rarely they are atrophied. The entire organ has a stony hardness. The cortical portion is of normal size or thickened, rarely thinned. It is markedly congested, either in the regular red streaks which normally mark the cortex, or, in extreme congestion, the entire cortex is dark red. The pyramids are congested, and of a dark-red color. The Malpighian bodies are usually unchanged, but filled with blood. The epithelium of the convoluted tubes is unchanged, except that it has a peculiar, stiff appearance. The veins are sometimes dilated.

This condition of chronic congestion may pass into chronic diffuse nephritis, and the appearance of the kidney will undergo corresponding changes.

INFLAMMATION.

Acute diffuse Nephritis.

This condition of the kidneys is found with scarlatina, and as an acute disease after exposure to cold. It is usually described as the first stage of chronic diffuse nephritis or Bright's Disease; in my opinion, improperly. It has been described by different authors under the names of interstitial nephritis, acute desquamative nephritis, acute Bright's Disease, croupous nephritis, etc.

The clinical history of most of these cases is: Pain in the back, fever, œdema of the face and legs; the urine scanty, high-colored; containing blood, albumen, renal epithelium; hyaline, granular, blood, and epithelial casts. The patient may die in a short time, but much more frequently entirely recovers. It is only exceptionally that this condition is succeeded by chronic nephritis. The kidneys are swollen; the capsule is not adherent; their texture is somewhat succulent. The entire organ is red and congested; or the cortex is white or gray, mottled with red spots; the pyramids are red. The tubes of the cortex and pyramids are more or less distended, with detached swollen or granular epithelium, with hyaline cast matter, and sometimes with blood. The Malpighian bodies are sometimes filled with extravasated blood. The intertubular connective tissue is infiltrated with a variable amount of serum and lymphoid cells.

Glomerulo-Nephritis.—Under this name, Klebs describes a modification of the condition just described. It is found with scarlatina. The Malpighian bodies appear large and white. Their capsules are filled with small cells, which compress the capillary tufts.

Chronic diffuse Nephritis.

This disease is also known under the names of parenchymatous nephritis, Bright's Disease, granular kidney, fatty kidney, etc.

It is the ordinary form of chronic renal disease, with which

all practitioners are familiar; attended during life with albumen and casts in the urine, with changes in the quantity and composition of the urine, with œdema of the face and legs, with various other symptoms, and ending, sooner or later, in the death of the patient.

The disease begins, for the most part, very slowly and insidiously. Very often it is impossible to fix the precise date of its commencement. It has no acute stage, but begins as a chronic change. Less frequently, it succeeds acute diffuse nephritis and chronic congestion of the kidney.

The gross changes in the kidneys affected by this disease are widely different; the minute changes are much more constant. The kidneys are of normal size, are enlarged, or are atrophied.

The kidneys, which are of normal size, are but little changed in appearance, except that the cortical markings are irregular, and the capsule is adherent; or the surface is slightly nodular; or the cortex is white, or grayish, or mottled white, gray, yellow, and red, and the organ looks as if it were atrophied from a previous condition of enlargement.

The large kidneys present their cortices of a white, gray, yellow, or mottled color. Their surfaces are nodular; their capsules adherent. The cortices are thickened; the pyramids are enlarged, red, and at their bases radiate in red and white streaks.

The atrophied kidneys exhibit all degrees of smallness. Both the cortex and pyramids are diminished in size. Their surfaces are nodular, and their capsules adherent. The fat about the pelvis is sometimes increased, so that the kidneys seem to be atrophied from within. The cortex is red, white, gray, or yellow, in a great variety of combinations and shades. The texture of the organ is firm. There may be cysts of various sizes throughout the kidney.

The minute changes in chronic diffuse nephritis are present in variable relative proportion, and in variable degrees of development.

The tubes are usually, but not always, dilated both in the cortex and pyramids. The dilatation may be slight, or so great as to form cysts visible with the naked eye. In the atro-

phied kidneys, there may be regions where the tubes are collapsed and atrophied. The basement membrane of the tubes may be thickened.

The epithelium of the tubes may be slightly swollen and granular; or very granular, broken down, and detached; or filled with oil globules; or, in the dilated tubes, flattened against the basement membrane.

Hyaline cast material, white, yellow, or mixed with broken epithelium, is usually present in the tubes, especially near the apices of the pyramids.

The arteries have their walls thickened, their cavities narrowed. It is usually impossible to inject them completely. Their walls may be the seat of waxy degeneration. The veins can usually be completely injected from the renal vein. The capillary venous plexus of the cortex may be dilated.

The Malpighian bodies are unchanged; or they are atrophied, and their capsules thickened; or the tufts are shrunk and impervious; or they are the seat of waxy degeneration.

There are usually changes in the intertubular tissue, especially in that of the cortex. We find just beneath the capsule, around the Malpighian bodies, and in other positions, irregular patches of connective tissue-fibres, and small round and oval cells. In the smallest and hardest kidneys, the cells may be as abundant in proportion to the fibrous tissue as in kidneys apparently in the early stages of the disease.

In the atrophied kidneys of gouty persons, we often find white lines and dots of urate of soda in the pyramids.

Pyelo-nephritis.

This form of kidney-disease runs an acute course, sometimes of only a few days, and is very fatal. The lesion of the kidneys alone is sufficient to destroy life.

The causation of this form of nephritis is peculiar, and hard to understand. It is secondary to inflammation of the pelvis, ureters, and bladder. But the inflammation may be confined to the bladder, and skip the ureters and pelvis, so that the infection appears metastatic rather than continuous. Some authors

explain this by the presence of fungi, bacteria, which find their way up the ureters, and into the kidney-tissue.

The lesions of the kidney are marked. The organ is somewhat swollen. On its surface, in the cortex, and in the pyramids, we find small white spots, surrounded by red zones of congestion. These spots may be from the size of a pin's head to that of a third of the kidney. They may be firm, or soft and purulent. The larger spots are usually purulent. The entire kidney may thus be converted into a bag of pus and broken-down tissue.

The minute change consists in an accumulation of lymphoid cells in the intertubular tissue, with degenerative changes in the tubes.

Perinephritis.

As a very rare occurrence, we sometimes find small collections of pus between the capsule and the kidney, apparently produced by inflammation of the capsule.

More frequently we find the products of inflammation outside the capsule. There may be, as the result of perinephritis, a mass of dense fibrous tissue surrounding the organ; or, more frequently, a collection of pus. These abscesses may open into the peritoneal cavity, into the intestine, or burrow in the muscles of the back, and open externally.

Perinephritis may be produced by blows on the lumbar region; by pyelitis and pyelo-nephritis; by abscesses in the psoas muscles; by abscesses in the pelvic cavity, and occur as an idiopathic inflammation after exposure to cold, and with typhus and other severe diseases.

DEGENERATIONS.

Chronic Parenchymatous Degeneration.

This disease is also known under the names of catarrhal nephritis, parenchymatous inflammation, granular degeneration, fatty degeneration.

It is found, not as a primary disease, but as a result of pro-

found changes in the nutrition of the body. Typhus fever, cholera, yellow fever, phthisis, chronic alcoholism, the acute exanthemata, phosphorus poisoning, and causes of like character, are the conditions with which this lesion is usually found. The urine contains albumen and granular casts. If the patient recovers from the acute disease, the kidney symptoms also disappear. The gross appearances of the kidney vary with the degree of degeneration which has taken place.

The kidney may be of normal size and appearance, the cortical markings regular. Or, it may be somewhat enlarged, the cortex opaque and white. Or, less frequently, the organ is congested. As a rule the capsule is not adherent, and the surface of the kidney is smooth.

The minute changes consist in an alteration of the epithelium in the tubes of the cortex, of the pyramids, or of both. In the lesser degrees of the lesion we find the epithelium swollen so as to fill the tubes, and at the same time more distinct and sharply outlined. In some of the tubes the epithelium is more granular, broken-down, and detached from the wall of the tube.

In other cases, the epithelium is more markedly granular, even fatty, and fills many of the tubes. There may be hyaline cast matter in the tubes of the pyramids. There are no changes in the intertubular tissue.

THE WAXY KIDNEY.

It has already been mentioned that, in chronic diffuse nephritis, the Malpighian tufts may be waxy. This change may also involve the small arteries, the veins in the pyramids, and even the basement membranes of the tubes.

We find various views, among different authors, as to the relationship between the waxy change in the vessels and the other changes in the kidney. Some hold that the condition is only an accidental complication of diffuse nephritis; others that the waxy degeneration is the first and essential part of the process, and that the other kidney lesions are secondary to it. I have never seen waxy degeneration of the tufts and vessels except in the advanced stages of chronic diffuse nephritis.

Fehr gives the following figures as representing the causation of the disease: Syphilis, 34; pulmonary phthisis, 32; with caries, 5; with other complications, 6; caries, with serofula, 26; empyema, 4; chronic bronchitis, 3; chronic alcoholism, 5; chronic articular rheumatism, 3; cancer, 3; intermittent fever, 4; hydronephrosis, 3; ulcers of the leg, 3; without known cause, 9.

INFARCTIONS OF THE KIDNEY.

Infarctions of the kidney are produced by the presence of an embolus in an artery or capillary vessel.

If the embolus is in an artery, the size of the infarction will correspond to the size of the vessel. As soon as the artery is plugged by the embolus, its branches are emptied or filled with thrombi. The wedge-shaped portion of kidney tissue, which is deprived of its supply of arterial blood, becomes of a deep yellow color, and is a little swollen, while the epithelium in its tubes degenerates. Afterward, the epithelium becomes more granular and disappears; the tubes become collapsed and shrunken. At the periphery of the infarction there is a zone of congestion, and of accumulation of lymphoid elements in the intertubular tissue. After this, the infarction may continue to shrink, and the zone of congestion around it helps in the formation of a fibrous cicatrix; or the production of lymphoid elements may be so great as to form an abscess; or the infarction becomes necrotic and gangrenous.

Capillary emboli produce a number of small infarctions. The kidneys are swollen, soft, mottled with numerous small, red spots, each with a white or yellow centre. At the centre of these hæmorrhagic spots there can generally be found a small vessel plugged with a thrombus. Around this the tubal epithelium is degenerated, and the intertubular tissue infiltrated with lymphoid cells. These latter cells may be so numerous as to form small abscesses.

The whole appearance may resemble very closely that of pyelo-nephritis.

Cohnheim has reported a case of complete embolism of the

renal artery, with necrosis of the entire kidney. It occurred in a boy eight years old, who was recovering from membranous croup of the larynx and trachea. The left ventricle of the heart contained a firm, white, laminated thrombus. The spleen contained a large, recent infarction. There were emboli in the left hypogastric artery, and in the right common iliac. The left renal artery and its branches were completely filled with a firm thrombus. The left kidney was somewhat increased in size. Its surface was smooth, mottled red and yellow. The cortex was dry, opaque, and looked like dead tissue. The pyramids were bluish red. Here and there in the cortex and pyramids were deep red spots.

Thrombosis of the renal vein occurs in consequence of changes in the kidney; from tumors obstructing the circulation; as a continuation of thrombi of the vena cava; and as a primary lesion dependent upon the general condition of the patient. Children, who are exhausted by diarrhœas, etc., are perhaps the most frequent subjects of this lesion. The thrombus may completely or only partially fill the renal vein and its branches.

HYDRO-NEPHROSIS.

Dilatation of the ureters and pelves of the kidneys is not uncommon. It is found at all ages, even before birth. It is produced by some mechanical obstruction to the outflow of urine. The obstruction may affect the pelvis, the ureter, the bladder, or the urethra. Calculi in the pelvis, or ureter; strictures of the ureters; new growths compressing the ureters, especially cancer of the uterus; changes in the bladder; strictures of the urethra are the ordinary causes.

The dilatation usually affects the ureter and pelvis—sometimes the pelvis alone, sometimes a single calyx. The pelvis and calyces are enlarged, the papillæ are flattened or converted into cavities until there is left a large multilocular cyst, of which the walls are formed of a thin layer of kidney tissue. The kidney substance which is left, although thinned, is not necessarily diseased. The epithelium may be entirely normal. In other cases, the epithelium may undergo granular degenera-

tion, or there may be chronic diffuse nephritis, or suppurative pyelo-nephritis.

THE CYSTIC KIDNEY.

We find cysts in the kidney in intra-uterine life, and at all ages.

The congenital cystic kidney is a very striking pathological condition. The kidneys are converted into a mass of numberless cysts, so large as to nearly fill the abdominal cavity, and often to seriously hinder the birth of the child.

The cysts are of all sizes, separated by fibrous septa, or by compressed kidney tissue. They contain a clear, yellow, acid fluid holding in solution the urinary salts. In other cases, the fluid is turbid and brown, and contains blood, uric acid crystals, or cholesterine. The cysts are lined with a simple layer of flat, polygonal epithelium. The cysts are formed by a dilatation of the kidney tubules, and of the capsules of the Malpighian tufts. The dilatation may be produced by a change in the papillæ, obliterating the tubes there; or by stenosis of the pelvis, ureters, bladder, or urethra. This condition is usually associated with other malformations, hydrocephalus, club-foot, etc.

In extra-uterine life, we find cysts in kidneys which are otherwise normal, and in those which are the seat of chronic diffuse nephritis.

In the normal kidney we find one or a few cysts, from the size of a pin's head to that of a pigeon's egg. They are situated in the cortex, and may project outward. They contain clear serum, or a viscid, colloid substance, or cholesterine. The walls of the cysts are smooth, or incomplete septa project inward. There is no exact knowledge of the way in which these cysts are formed, although it is probable that they are produced by the dilatation of the kidney tubules. They do not give rise to any changes in the kidney tissue, nor to any clinical symptoms.

In kidneys which are the seat of chronic diffuse nephritis it is not infrequent to find cysts. The cysts are numerous, scat-

tered through the cortex and pyramids, usually small, not often larger than a small chestnut. The cysts contain clear serum, or colloid matter, or brownish pigment. Their walls are thin, smooth, and lined with pavement epithelium. The larger cysts are formed by the fusion of several smaller cysts. In some of the cysts the remains of the atrophied Malpighian tufts can be found. The kidneys are usually atrophied, sometimes large. Minute examination shows the lesions of diffuse nephritis, with dilatation of the tubes and Malpighian capsules. In many kidneys, all the different stages of the dilatation process can be distinctly seen. The dilatation does not seem to be always due to compression of the tubes by new fibrous interstitial tissue; but is also caused by the plugging of the tubes with colloid cast-matter.

PRECIPITATES AND CONCRETIONS.

The Urates.—In the kidneys of new-born children, we often find narrow, orange-colored lines in the pyramids parallel to the tubes. These lines consist of small, brownish-yellow, rounded, or angular bodies, resembling the sediment of urate of ammonia so often found in the urine. If acetic acid is added, these bodies are dissolved, and crystals of uric acid are formed. These deposits are found, as a rule, in infants which have breathed from thirty-six to forty-eight hours. Exceptional cases have been reported, however, in which the same condition was found in still-born children.

Pigment.—In new-born children there are sometimes reddish-brown lines in the papillæ and pyramids. They are composed of granules of pigments and crystals of hæmatoidine. They are the results of small hæmorrhages in the tubes. They can be distinguished from the deposits of urates by the chemical reaction.

The Carbonate and Phosphate of Lime may be deposited in the tubes of the pyramids, in the form of white lines. They are found in old persons, and with destructive diseases of the bones.

Urate of Soda is deposited in white lines and spots in the

pyramids and cortex in some cases of chronic diffuse nephritis, especially in gouty individuals. It is deposited both in the tubes and in the intertubular tissue, in the form of acicular crystals.

Concretions of the urinary salts of all sizes and shapes, and in indefinite numbers, may be formed in the calyces and pelvis of the kidney. They may pass through the ureter into the bladder; or cause hydro-nephrosis, or pyelo-nephritis.

NEW GROWTHS.

Fibroma.—Small, hard, white, fibrous nodules are frequently found in the pyramids. The kidneys are normal, or the seat of diffuse nephritis.

Myxoma.—Small nodules of mucous tissue are sometimes found in pyramids. Large tumors may grow from the pelvis of the kidney. They are usually a combination of myxoma, lipoma, and sarcoma.

Lipoma.—There may be a diffused increase of fatty tissue around the capsule of the kidney; and in atrophied kidneys an increase of fat about their pelves. Small, rounded, or lobulated, fatty tumors are occasionally found in the cortex, just beneath the capsule.

Tubercles.—With general tuberculosis we often find small, miliary, gray, or cheesy granulations scattered through the kidney.

Besides this condition, there is another change in the kidney, which is generally called tubercle, but of which the real nature is somewhat doubtful. First the papillæ, and afterward the rest of the pyramids and the cortex, are converted into a yellow, cheesy mass, of firm or soft consistence. The entire kidney tissue may be destroyed in this way. The pelvis and the ureter are usually coated with a thick layer of the same cheesy material. Similar changes in the bladder, vesiculæ seminales, testicle, prostate, uterus, and Fallopian tubes often exist in the same patient. The lesion is usually seen at such an advanced stage that nothing but the cheesy degeneration can be found. It is said that this condition is preceded by the formation of miliary

tubercles, which become aggregated and form the cheesy mass.

Leukæmic Tumors are sometimes found in the kidneys in connection with similar growths in the other viscera. The tumors are small, whitish, composed of lymphoid cells, and situated in the intertubular tissue.

Syphilitic Gummata have been observed in a few cases. They were in the form of nodules of the usual character. Klebs describes a more diffuse growth of fusiform cells between the tubes in a case of congenital syphilis.

Sarcoma occurs in the kidney in the form of metastatic nodules. Myxo-sarcomata grow from the pelvis.

Adenoma.—Klebs describes under this name tumors of small size, situated in the cortex. They are white, soft, and rounded. They consist of a connective tissue stroma, supplied with vessels, and in which are long, tubular, anastomosing spaces filled with small, polygonal, epithelial cells. The relationship of this form of tumor to true carcinoma is not well established.

Carcinoma occurs in the kidney as a primary or secondary disease. The secondary tumors are usually of moderate size, and may occur in both kidneys.

Primary carcinoma usually affects one kidney—more frequently the right than the left. It is most frequently of a soft, medullary, hæmorrhagic character. But scirrhus, melanotic, and colloid cancer also occur. The tumors gradually replace the kidney tissue, and often reach an enormous size. The new growth may project into the ureters, into the renal vein, and through this into the vena cava.

Angioma Cavemosum occurs in the form of small, encapsuled nodules, situated in the cortex. Their structure is the same as that of the angiomata of the liver.

PARASITES.

Echinococci are of not infrequent occurrence in the kidney.

Cysticercus cellulosæ and *Pentastoma denticulatum* have been seen in a few cases.

Eustrongylus gigas is sometimes found in the pelvis of the kidney.

THE URINARY BLADDER.

MALFORMATIONS.

Ecstrophia, Extroversion, Inversion, Fissure of the Bladder is one of the most frequent malformations. It occurs in both sexes.

(1.) The umbilicus is lower down than usual, the pubic bones are not united at the symphysis, the pelvis is wider and shallower than it should be.

Between the umbilicus and pubes the abdominal wall is wanting. In its place is a projecting ovoid mass of mucous membrane, in which can be seen the openings of the ureters.

The penis is usually rudimentary; the urethra is an open fissure, (epispadia;) the clitoris may be separated into two halves. The ureters usually open normally; sometimes their openings are displaced, or are multiple. They may be dilated.

(2.) Vrolik describes a case in which there was a fissure in the abdominal wall, which was filled up by the perfectly formed bladder.

(3.) The umbilicus may be well formed, and there is a portion of abdominal wall between it and the extrophied bladder.

(4.) The external genitals and urethra may be well formed, and the symphysis pubis united, while only the bladder is fissured.

(5.) The genitals, urethra, and symphysis may be well formed, the bladder closed, except at the upper part of its anterior wall. The bladder is entirely or in part inverted and pushed through the opening in the abdominal wall.

The Urachus normally remains as a very small canal, 5-7 centim. long, with a small opening into the bladder, or entirely closed at that point. If there is a congenital obstruction to the flow of urine through the urethra, the urachus may remain open, and the urine pass through it.

Absence of the Bladder is very rare, and only described by the older authors.

The bladder may be very small, and the urine pass almost directly into the urethra.

The bladder may be separated into an upper and a lower portion by a circular constriction. It may be completely or incompletely divided by a vertical septum into two lateral portions.

Diverticula of the wall are also found in new-born children.

Partial or complete closure of the neck of the bladder also occurs.

CHANGES IN SIZE AND POSITION.

Dilatation of the bladder is produced by the accumulation of urine in consequence of some mechanical obstacle to its escape, or of paralysis of its muscular walls. The dilatation is uniform, and may reach a great size. If the walls of the bladder are paralyzed, the dilatation is accompanied by thinning of the wall, or it may retain its normal thickness. If the escape of the urine is prevented by a mechanical obstacle, the walls of the bladder are usually hypertrophied, but may be thinned. The retained urine frequently decomposes, and produces inflammatory and gangrenous changes in the mucous membrane.

In the foetus, dilatation of the bladder may reach such a size as to interfere with delivery.

I have seen, in a foetus born at term, dilatation and hypertrophy of the bladder, dilatation of the ureters, a diverticulum at the neck of the bladder, and no apparent obstacle to the escape of urine.

Diverticula of the bladder are produced by the protrusion of the mucous coat between the hypertrophied bundles of muscular fibres. They may be single or multiple, small, or so large as nearly to fill the abdomen. Their walls are composed of mucous membrane thickened, and of peritoneum, if in the proper position. They communicate with the bladder by a small cleft. The urine which fills them may decompose and excite inflammation. Calculi may be formed in them.

Hypertrophy of the muscular coat of the bladder is usually produced by mechanical obstructions to the outflow of urine, such as stricture of the urethra, enlarged prostate, calculi, new growths, etc. The muscular coat is thickened uniformly, or

assumes a trabeculated appearance. The organ retains its normal capacity, or is dilated, or becomes smaller. The mucous membrane is frequently the seat of chronic or acute inflammation. Dilatation of the ureters and hydronephrosis frequently accompany this condition.

Herniæ of the bladder sometimes accompany intestinal herniæ through the inguinal and crural canals, and the foramen ovale. The changes in position of the bladder, produced by displacements of the vagina and uterus, will be mentioned with the lesions of those organs.

RUPTURE.—PERFORATION.

Penetrating wounds of the bladder may produce escape of urine into the abdominal cavity, or infiltration into the surrounding connective tissue, or permanent fistulæ. Such wounds are always serious and frequently fatal.

Rupture of the bladder may be produced by severe blows and falls when the bladder contains urine. More rarely, rupture takes place from over-distention. I have seen this occur in a patient who was being etherized preparatory to puncturing the bladder. Fractures of the pelvic bones may produce laceration of the bladder.

Perforations of the bladder are produced by ulceration and gangrene, by abscesses from without, and by cancerous ulceration from the adjoining organs.

INFLAMMATION.

Catarrhal Cystitis may be idiopathic; or excited by decomposed urine, or by calculi; or be an extension of gonorrhœal urethritis, or vaginitis. It may run an acute or a chronic course.

In the acute form, the mucous membrane is swollen and intensely congested. Pus, mucus, and blood are set free from its surface.

In the chronic form, the mucous membrane is at first swollen, succulent, grayish, or mottled with spots of congestion and extravasation, covered with a layer of muco-pus. Later, the

mucous membrane becomes brown, or slate-colored, its swelling is more marked, and may take the form of tufts or polypi. The submucous and muscular coats are hypertrophied. The mucous membrane may be incrustated with the salts of the decomposed urine. Ulcerations take place which gradually perforate all the coats of the bladder and produce abscesses in the surrounding connective tissue. These abscesses may finally open into the vagina, intestines, or through the skin. In other cases, the muscular coat is paralyzed, and the bladder dilated. The mucous membrane is discolored, incrustated with salts, gangrenous. The gangrene may extend to all the coats, and the urine escape into the abdominal cavity. In all cases the inflammation may extend to the ureters and kidneys.

Croupous and Diphtheritic Inflammation may accompany the same inflammation of the vagina; occur with scarlet fever, typhus fever, etc., with stone in the bladder and decomposing urine, with carcinoma; and are rarely an idiopathic disease. The entire mucous membrane or only patches of it are involved. It is intensely congested, or of a grayish color, covered and infiltrated with fibrine and pus, portions of it gangrenous. Sometimes entire fibrinous casts of the bladder are discharged by the urethra; very rarely the mucous membrane itself is detached and cast off.

NEW GROWTHS.

Fibroma, in the form of circumscribed nodules in the submucous tissue, is described by Rokitsansky as of very rare occurrence.

Fibroma papillare occurs in the form of a mass of branching tufts, covered with cylindrical epithelium. The muscular coat of the bladder is often hypertrophied at the same time. The growth is most frequent in young persons, and even occurs in children.

Fibroma polyposum occurs in the form of a circumscribed hypertrophy of the mucous membrane, usually near the neck of the bladder. It is most frequent in childhood.

Fibro-Adenoma is found at the neck of the bladder, in the sub-

mucous tissue, or projecting inward in a polypoid form. The tumors are from the size of a pea to that of a pigeon's egg. They are composed of a fibrous stroma and of glandular follicles, resembling those of the prostate. They seem to be detached outgrowths from the prostate.

Enchondroma.—A somewhat doubtful case of diffuse infiltration of the entire wall of the bladder with cartilage is described by Ordóñez.

Tubercles of the bladder are sometimes found in connection with tubercles of the testes, prostate, vesiculæ seminales, and kidneys. They are only found in the male. There are at first gray granulations in the superficial layers of the mucous membrane. These soften and form ulcers, of which the walls are infiltrated with new granulations. In this way large ulcers with cheesy floors and edges are formed.

Cysts.—Paget mentions a case of a dermoid cyst in the wall of the bladder. Sometimes small serous cysts are found in the mucous membrane.

Carcinoma of the bladder is most frequently secondary to the same growth in neighboring organs.

As a primary growth it occurs:

(1.) As a diffuse scirrhus infiltration of the entire wall of the bladder, usually with ulcerations of its inner surface.

(2.) As a circumscribed nodule which grows inward and outward, ulcerating on its inner surface, and sometimes producing perforations.

(3.) As villous or epithelial cancer. The tumor grows from one or more points of the inner surface of the bladder. It is formed of tubular follicles, lined with cylindrical epithelium, and on its inner free surface of tufts covered with cylindrical epithelium. The new growth may involve the entire thickness of the wall of the bladder.

(4.) As myo-carcinoma. Virchow describes a tumor of this kind situated at the trigonum, and composed of fibrous and smooth muscular tissues, forming cavities filled with epithelial cells.

I have seen one such case, in which there were two small tumors at the neck of the bladder. They were composed of a

fibrous and muscular stroma, forming cavities filled with epithelial cells. The tumors were softer, and the muscular tissue more abundant, than in the case described by Virchow.

PARASITES.

Distoma hematobium has been observed in the vessels of the mucous membrane—only in Egypt.

Sarcina has been found with chronic cystitis.

Eustrongylus, *echinococcus*, and *ascarides* sometimes find their way into the bladder from other organs.

Every variety of foreign body may be introduced into and remain in the bladder. If their stay is long, they become incrustated with the urinary salts.

THE URETHRA.

CONGENITAL MALFORMATIONS.

Some of the malformations of the urethra are described with those of the penis.

The urethra may be impervious, or may open at the root of the penis. More commonly there is partial obliteration or stricture of some part of the canal.

The entire urethra may be dilated into a sac full of urine.

There may be a canal on the dorsum of the penis, formed by the fusion of the spermatic cords, and opening in the glans above the urethra.

There may be two or more openings of the urethra.

The canal may be dislocated so as to open in the inguinal region.

CHANGES IN SIZE AND POSITION.

Dilatation of the urethra is produced by strictures, or by calculi which become fixed in it. The dilatations are fusiform or sacculated in shape, and may reach the size of an orange and over.

Strictures of the urethra are produced by inflammation of its walls.

The stricture may be temporary, produced by a diffused inflammatory swelling of the mucous membrane, or by the raising of the relaxed membrane into a fold or pocket.

Permanent strictures are produced by structural changes in the walls of the urethra.

(1.) The mucous membrane and sub-mucous tissue is left hard and unyielding by the preceding inflammation. Subsequently, the new fibrous tissue contracts and narrows the canal.

(2.) Ulceration of the mucous membrane leaves cicatricial tissue, which contracts, and also produces adhesions and bands of fibrous tissue.

(3.) There is fibrous induration of the corpus spongiosum, and consequent constriction of the urethra.

The most frequent position of strictures is at the junction of the membranous and spongy portions of the urethra, or close to this point. They also occur at the fossa navicularis and the meatus, but frequently in the prostatic portion. There may be one stricture or several.

The consequences of stricture are dilatation of the urethra, the bladder, the ureters, and hydronephrosis; inflammation and ulceration of the urethra behind the stricture, with perforation, infiltration of urine, or the formation of fistulæ.

The urethra may also be obstructed by folds of the mucous membrane; by muscular valves at the neck of the bladder; by wounds; by polypi and swollen glands; by new growths; by changes in the prostate and perineum; by calculi, mucus, blood, and echinococci coming from the bladder; by foreign bodies introduced from without.

Prolapse and inversion of the mucous membrane is seen in young girls and women in rare cases. There is a bluish, red swelling, from the size of a pea to that of a walnut, at the meatus. In the male, invagination of the mucous membrane of the urethra has been seen after injuries of the perineum.

WOUNDS, RUPTURE, PERFORATION.

Wounds of the urethra are produced in many ways, but most commonly by catheters and bougies. The wounds may cicatrize,

or there may be infiltration of urine, or the formation of fistulæ.

Ruptures of the urethra are produced by severe contusions and by fracture of the pelvic bones. Extravasations of blood and urine, and gangrenous inflammation of the surrounding soft parts, are the ordinary results.

Ulceration and perforation of the urethra lead to the formation of fistulæ, which open in various directions through the skin.

INFLAMMATION.

Cutarrhal Urethritis is most frequently due to infection from gonorrhœa, but is also produced by a variety of causes. In its acute form, it involves either a portion or the whole of the urethra. The mucous membrane is red, swollen, and covered with muco-pus. The inflammation may extend to the fibrous wall of the urethra, the corpora spongiosa, and cavernosa. This may result in the formation of new connective tissue, or of abscesses, especially near the fossa navicularis. The inflammation may also extend to the bladder, the glands of Cowper, the prostate, the spermatic cord, and the testicles. The inguinal glands also may be swollen and inflamed, and the lymphatic vessels on the dorsum of the penis may be involved in the same process.

Chronic inflammation of the urethra may exist for a long time without producing any lesions. In other cases it leads to ulceration, to fibrous induration of the wall of the canal, to induration and swelling of the mucous follicles; to polypoid thickenings of the mucous membrane.

Croupous Inflammation is sometimes seen in children. Fibrinous casts of a small or large portion of the canal are formed.

Syphilitic Ulcers may be situated at the meatus, or as far back as the fossa navicularis. They are apt to produce strictures.

NEW GROWTHS.

Fibroma Papillare, in the form of little polypoid excrescences of the mucous membrane, sometimes occurs. They are the result of chronic inflammation, and are confined to a small or large portion of the wall of the urethra.

Tubercles are rarely found in the mucous membrane of the urethra in connection with tubercles of the bladder, prostate, or testicles.

Carcinoma is usually only a part of some new growth of the penis, prostate, or bladder. Very rarely, isolated nodules are found in the wall of the urethra, secondary to carcinoma of the bladder.

THE ORGANS OF GENERATION.

THE VULVA.

MALFORMATIONS.

THE external genitals may be entirely absent, or imperfectly developed.

The clitoris and nymphæ may be abnormally large, or the nymphæ may be increased in number.

The clitoris may be abnormally long, resembling a penis, at the same time the vagina is narrow, the uterus small and undeveloped, the ovaries small, sometimes situated in the labia, the mammæ small, and the body of a masculine character.

The clitoris may be perforated by the urethra, or may be cleft and apparently double.

The large or small labia may grow together, with or without obstruction of the urethra. There may be a fleshy septum above the lower commissure of the vulva.

HÆMORRHAGE.

Hæmorrhage may take place from wounds or ulcers of the vulva.

The most important form of hæmorrhage is that which occurs in the connective tissue of the labia majora. This is produced during labor. One of the labia may be swollen and distended by the extravasated blood, until it is as large as a child's head. The blood may be gradually absorbed, or it may become gangrenous with suppuration of the surrounding tissue. The purulent matter may escape through the skin, and the patient recover; or the suppuration may extend into the pelvis, and cause death.

INFLAMMATION.

The skin, mucous membrane, connective tissue, and glands of the vulva may be the seat of inflammation.

Chronic catarrhal inflammation may produce a thickening and papillary hypertrophy of the cutis, with a moist ulcerating surface.

Erysipelatous inflammation of the skin of the vulva is frequent in young children, and may cause death. In adults, it is less common.

Acute œdema of the labia majora occurs in pregnant and puerperal women. It may terminate in gangrene or in suppuration.

Inflammation of the vulvo-vaginal glands may be acute, and produce abscesses; or chronic, and produce induration of the gland.

Gangrene may follow erysipelatous inflammation, may occur after parturition, may accompany severe and exhausting diseases, and may occur as an epidemic disease, especially among children.

Herpes, Eczema, Lichen, Prurigo, etc., may be found on the skin of the vulva.

NEW GROWTHS.

Fibroma.—*Fibroma diffusum* (elephantiasis) is found growing from the clitoris, the nymphæ, and the labia majora. It appears as a diffused hypertrophy of the parts, with a smooth or warty surface; or as a polypoid growth with a narrow pedicle. In either case the structure is the same. If the skin remains smooth, its outer layers are but little changed; if it is rough, the papillæ are hypertrophied. The new growth arises from the deep layers of the cutis, and consists of a loose connective tissue infiltrated with serum. The tumors may reach a very large size.

Fibroma Papillare, Papilloma.—These tumors vary from the size of a pea to that of an apple. They present a rough, cauliflower-looking appearance. They are composed of papillæ or tufts, with a central stroma of connective tissue and blood-vessels and a thick covering of epithelium.

Fibroma Tuberosum.—Circumscribed fibrous tumors are found in the connective tissues of the labia, the mons veneris, the perineum, the clitoris, and the entrance of the vagina. They may attain a large size, and may be attached only by a narrow pedicle. When a section is made, it will be found that the tumor is a circumscribed growth, not continuous with the skin.

Lipoma.—Fatty tumors are found in the mons veneris and labia majora. They may reach a large size.

Enchondroma.—Schneevoigt describes a polypoid, cartilaginous tumor growing from the clitoris.

Cysts are found in the connective tissue of the labia majora and minora. They are from the size of a pea to that of a child's head. They contain serum, colloid matter, pus, or blood, or have the character of dermoid cysts. They may open through the skin. Cysts are also formed by dilatation of the ducts or acini of the vulvo-vaginal gland.

Carcinoma of the vulva is rare. It is usually accompanied by the same growth in the uterus and vagina, or in the other viscera. It occurs in the form of nodules, which, after a time, ulcerate.

Epithelioma is more common. It usually begins in one of the labia majora. There is at first induration and thickening of a circumscribed portion of the labium, afterward ulceration. The inguinal glands may become involved, but metastatic tumors are rare.

Lupus occurs in the labia majora and minora, the mons veneris, and the perineum in its usual forms. The cutis is infiltrated with small round cells. When the cell-growth reaches the surface of the skin, the epithelium falls off, and an ulcerating surface is formed. According to the extent of the infiltration and of the ulceration, we have the varieties: *Lupus maculosus*, *nodosus*, *hypertrophicus*, *exulcerans*.

THE VAGINA.

MALFORMATIONS.

The vagina may be entirely absent, and the internal organs of generation also absent, or imperfectly developed.

Either the upper or the lower portion of the canal may be absent while the remaining portion is present.

The vagina may be closed by an imperforate hymen, or by fibrous septa at any part of its canal. The canal may be abnormally small without being occluded.

The vagina may be double, in connection with a double uterus; or, while the uterus is normal, the vagina may be incompletely divided by a longitudinal septum.

The hymen is seldom entirely absent, but is often either too large or too small.

CHANGES IN SIZE AND POSITION.

Dilatation of the vagina is produced by tumors, by the prolapsed uterus, and by the accumulation of blood and mucus behind constrictions or obliterations of the canal.

Lengthening of the vagina is produced by any cause which draws the uterus upward.

Narrowing of the vagina is found as a senile change; is produced by tumors, and by ulceration of the wall of the canal. Extensive ulcers may even cause entire obliteration of the canal.

Prolapse of the vagina occurs by itself or in connection with prolapse of the uterus. As an idiopathic process, it usually takes place soon after parturition. A larger or smaller portion of the canal is inverted and projects through the vulva. The entire circumference of the canal may be inverted and prolapsed, or only the anterior or posterior wall. The prolapse is at first small, but afterward gradually increases in size, and may drag down the uterus with it.

In other cases, prolapse of the uterus is the primary lesion, and the vagina is inverted by the descent of that organ; or, the body of the uterus may retain its normal position, while an

hypertrophy and lengthening of the cervix alone drags down the vagina.

Hernia vesico-vaginalis, Cystocele, may be either the cause or effect of a prolapse of the vagina and uterus.

If the cystocele is the primary lesion, it begins as a small projection of the wall of the bladder into the anterior part of the vagina. As the urine accumulates in this sac, it increases in size, projects through the vulva, draws down the vagina and the anterior lip of the cervix, finally the entire uterus.

If the cystocele is the secondary lesion, it is simply produced by the dragging down of the posterior wall of the bladder by the inverted vagina.

Hernia intestino-vaginalis.—A portion of the intestines may become fixed in Douglas's cul de sac between the rectum and uterus. This portion of intestine gradually becomes larger, pushes forward the posterior wall of the vagina, inverts and fills up that canal, and, finally, projects through the vulva. It may drag with it the posterior wall of the vagina and the uterus.

Rectocele vaginalis.—A sac is formed by the projection of the anterior wall of the rectum and the posterior wall of the vagina. This lesion is of rare occurrence, and does not reach a large size.

WOUNDS. PERFORATIONS.

Wounds of the vagina are made by penetrating instruments, by forceps and other obstetrical weapons, and by the fœtus during delivery. Such wounds may heal; may give rise to large hæmorrhages; may suppurate; may produce abscesses in the surrounding tissues; may leave fistulous openings into the vagina; or may cause constriction or obliteration of its canal.

Vesico-vaginal fistulæ are usually produced by injuries from instruments or from the fœtus during delivery; less frequently by ulceration of the vagina, bladder, or adjacent connective tissue. The fistulæ form an opening between either the bladder or the urethra and the vagina. They allow the urine to pass into the vagina. Spontaneous cure does not take place.

Recto-vaginal fistulæ are formed in the same way as the last

mentioned. They allow the passage of fæces into the vagina. They sometimes heal spontaneously.

INFLAMMATION.

Catarrhal Inflammation of the vaginal mucous membrane may be acute or chronic. It is caused by gonorrhœa, by local irritation, or depends on general causes. In the acute form, there is an abundant production of pus. In the chronic form, the mucous membrane may be thickened, rough, and covered with hard papillæ; or it may become relaxed and prolapsed.

Croupous and Diphtheritic Inflammation occur with general diseases and after parturition. The mucous membrane is covered with a grayish layer of fibrine and pus-cells, in which the salts of the urine may be deposited. The mucous membrane and the submucous tissue may also be infiltrated with fibrine and pus-cells. The gangrene and degeneration of these infiltrated portions of the mucous membrane may leave deep ulcers.

Suppurative Inflammation of the fibrous coat of the vagina is produced by injuries, and occurs in pregnant and puerperal women. Abscesses are formed, which may penetrate into the labia, or into the pelvic connective tissue.

Syphilitic Ulceration may be found in any part of the vagina.

NEW GROWTHS.

Myomata are sometimes found growing from the posterior wall of the vagina. They usually grow backward and upward, very rarely project into the cavity of the vagina in a polypoid form.

Papilloma.—Small and large polypoid growths composed of papillæ, or tufts covered with epithelium, are occasionally met with.

Tubercles of the vagina are rare. They accompany tuberculosis of the uterus, or of the urinary apparatus. They are found in the form of gray granulations, or of a diffused, yellow layer formed of many such granulations. •

Cysts are sometimes met with deep in the vaginal mucous

membrane. They are usually small, and contain serum or mucus.

Sarcomata are sometimes found in the wall of the vagina. They may surround the urethra.

Carcinoma is usually secondary to the same new growths in the uterus, but may be primary. It presents the same forms as are described in the uterus.

Myoma strio-cellulare myxomatodes.—Under this name Frau Kaschewarowa-Rudnewa has described a peculiar tumor of the vagina. The tumor occurred in a girl 15 years old. It grew from the anterior wall of the vagina, and first appeared like a small polypus. It was removed, but recurred in a few weeks. It then grew rapidly until it reached the size of a child's head. The patient died exhausted by the disease. The growth was composed of round, fusiform, and stellate cells imbedded in a mucous basement substance, and of striated muscular fibres.

THE UTERUS.

CONGENITAL MALFORMATION.

The uterus, up to the end of the third month of intra-uterine life, consists of two large cornua, which, by the fusion of their lower ends, form the uterus.

The uterus, tubes, and vagina may be entirely absent, with or without absence of the ovaries and external genitals. Or the uterus alone, or the upper part of the vagina also, may be absent.

The uterus may be only rudimentary, while the vagina is normal. It then appears as a flattened, solid body, with solid cornua. Or there are two cornua joined at their lower extremities so as to form a small double uterus. Or the uterus is represented by a small sac, which may or may not communicate with the vagina. Or there is a very small uterus with thin, muscular walls and two large cornua.

Only one of the cornua, which should form the uterus, may be developed, while the other is arrested in its growth. The uterus is then a long, cylindrical body, terminating above in

one tube. On the side where the other horn should have been developed, there is no tube, or only a rudimentary one. Both ovaries are usually present.

The two cornua may be fully developed, but their lower ends remain separated, and form a double uterus. An entire separation into two distinct uteri and vaginae is very rare. More frequently, the uterus consists of one body, divided by a septum into two cavities. There are then two cervical portions of the uterus projecting into a single vagina, or each into a separate vagina. Or there is only a single cervix. The septum in the uterus may be complete, or only partial.

We also find abnormal size of the uterus, abnormal flexions; the cervix may be solid, or may be closed by the vaginal mucous membrane.

CHANGES IN SIZE.

In the new-born infant, the uterus is small, the body flattened, the cervix disproportionately large. During childhood, the organ increases in size, but the body remains small in proportion to the cervix. At puberty, the shape changes, and the body becomes larger. Its length is then two to three inches; its breadth, sixteen to twenty lines; its thickness, eight to twelve lines. At every menstruation the uterus is somewhat swollen and congested. After pregnancy, it does not return to its virgin size, but remains somewhat larger. In old age, it gradually becomes smaller; its walls are harder and more fibrous.

Abnormal Smallness of the uterus is sometimes found as an arrest of development. The uterus in adult life retains the size and shape of that of the infant.

Atrophy of the uterus results from chronic endometritis, from repeated pregnancies, from puerperal endometritis, and from old age. Its walls are friable, its size is diminished, its cavity is smaller, or is distended with mucus. Large myomata sometimes cause marked atrophy of the uterine wall.

Atrophy of the vaginal portion of the uterus is sometimes observed after repeated pregnancies, sometimes without known cause.

Narrowing and obliteration of the cavity of the uterus and

of the cervix are usually produced by chronic inflammation. They may cause an accumulation of blood or mucus, and consequent distention of the uterus.

Hypertrophy of the uterus may occur in consequence of too early development. It is accompanied with abnormally early development of all the sexual organs and functions.

Heart-disease, prolapse and abnormal flexions and versions of the uterus, chronic inflammation, repeated pregnancies, myomata, accumulations of blood or mucus in the uterine cavity, may all produce hypertrophy of the uterus.

Hypertrophy of the vaginal portion is produced by the same causes as the above, and also is found without known cause. The hypertrophy consists in a uniform increase of size of one or both lips, or either one or both lips are hypertrophied and irregularly lobulated. There may be, at the same time, retention-cysts formed in the mucous follicles of the cervix.

Dilatation of the uterus is produced by accumulations of blood, mucus, or pus, in consequence of narrowing or obliteration of the cervix or vagina. The walls of the uterus may retain their normal thickness, be thickened or thinned.

The most frequent position of the stenosis is the os internum. The retained mucus and pus, after a time, change their character, and are replaced by a thin serum—hydrometra, or may be mixed with blood. The dilated uterus is not usually larger than an apple, but cases are recorded of enormous dilatation.

If both the os internum and os externum are closed, the cavity of the cervix is also dilated, and the uterus has the shape of an hour-glass.

If the obstruction is in the vagina, the uterus and vagina form a large flask-shaped body, and the line of demarcation between the cervix and vagina is lost.

In some cases, the dilatation is confined to the cervix.

If the obstruction is not complete, the retained fluid may escape into the vagina, and afterward accumulate again.

Hæmatometra.—Accumulation of menstrual blood in the cavity of the uterus is usually produced by congenital stenosis of the cervix or vagina. The dilated uterus may reach an enormous size. If the fluid is not evacuated by surgical interference,

there may be either rupture or ulcerative perforation of the uterus. The blood may escape into the abdominal cavity, or be shut in by adhesions, or perforate into the bladder or intestines. Sometimes the blood passes into the Fallopian tubes, dilates them, and escapes through their abdominal ends.

CHANGES IN POSITION.

The body of the uterus may become fixed in an abnormal position; while the situation of the cervix is unchanged. The body may be bent forward—ante-flexion; backward—retroflexion; or sideways—lateral flexion. The flexion may be slight, or so great that the neck and body form an acute angle. Ante-flexion is the most common variety, and that in which the flexion is greatest.

Peritoneal adhesions, flaccidity of the uterine walls, atrophy, ovarian tumors, etc., are the usual causes.

The Versions of the uterus consist in an abnormal inclination of the long axis of the organ to that of the vagina. The uterus may be inclined backward, forward, or to one side.

Retroversion is very much the most common. The fundus uteri is directed backward and downward; the cervix, forward and upward. This condition is found in various degrees; in the highest, the fundus lies in Douglas's cul de sac with the cervix upward, so that the axis of the uterus is parallel to that of the vagina, but in a direction exactly opposite to the normal one.

Abnormal looseness of the uterine ligaments, abnormally large capacity of the pelvis, hypertrophy or tumors of the uterus, and pregnancy during the first four months, are the usual causes of this lesion.

The greater degrees of version may produce very grave lesions. The urethra and rectum are compressed. Cystitis, perforation of the bladder, dilatation of the ureters and hydronephrosis, and fatal obstruction of the bowels may follow.

If pregnancy exist, abortion may take place, or the inverted uterus may be forced through the peritoneum and posterior wall of the vagina, and project through the vulva.

In the non-pregnant uterus, pressure on the veins, and consequent chronic inflammation of the organ, may follow.

Anteversion. Inclination of the fundus forward and downward, and of the cervix backward and upward, is not common, and seldom reaches a high degree. Pressure on the bladder and rectum may be produced by it.

Lateral version is produced by congenital shortness of one of the broad ligaments, by adhesions, or by the pressure of tumors.

Prolapsus Uteri consists in a descent of the uterus in the vagina. The position of the uterus may be only slightly lowered, or it may project through the vulva. In complete prolapse, we find a large tumor projecting through the vulva, covered by the distended vagina, and presenting the opening of the os externum near its centre. The bladder and rectum may be drawn down with the vagina, or may remain in place. The exposed cervix and vagina may become inflamed and ulcerated, or the mucous membrane may become thick and resistant.

The process is frequently complicated by hypertrophy of the cervix. In the lesser grades of prolapse, the hypertrophy of the cervix often seems to be the most important part of the lesion.

Sudden prolapse usually occurs after parturition, less frequently in virgins and in old women. Gradual prolapse is frequently caused by a cystocele or rectocele of the vagina. The vagina is dragged down, and draws the uterus with it.

Elevation of the uterus is produced by mechanical causes—adhesions, tumors, etc. The vagina is drawn up and lengthened, and the vaginal portion of the cervix is obliterated.

Inversion of the uterus consists in an invagination of the fundus. The fundus may be invaginated in the body; the fundus and body in the cervix; or the entire organ in the vagina. The most common cause is traction on the umbilical cord during parturition. It may also take place spontaneously after parturition. Lesser degrees of inversion are produced by intra-uterine polypi, or hydro- or hæmatometra.

Herniæ of the uterus are very rare. Ventral herniæ occur during the latter months of pregnancy. The peritoneum,

aponeuroses, and skin are forced outward to form a sac, in which the uterus is placed. Crural herniæ are produced by the drawing down of the uterus and ovaries into the sac of an intestinal hernia. Inguinal hernia is produced in the same way, or is congenital. Ischiadic hernia has been seen in one case.

Pregnancy may occur in the uterus while situated in a crural or inguinal hernia.

RUPTURE PERFORATION.

Rupture of the unimpregnated uterus is rare. It may, however, occur when the uterine cavity is distended with blood or serum, or in connection with large myomata of the uterine walls.

In the gravid uterus, ruptures have been seen in nearly every month of pregnancy. The rupture may be produced by thinning of the uterine wall by tumors, or by violent contusions.

The act of parturition is the most frequent cause. Malpositions of the fœtus, narrowing of the pelvis, long protracted labor, thinning of the uterine wall from tumors, forcible use of the forceps and other instruments are the ordinary causes. The rupture may be in the body of the uterus or the cervix; it may be large or small; it may extend completely or only partly through the uterine wall. The consequences of partial rupture are hæmorrhage, gangrenous inflammation of the edges of the rupture, peritonitis, and usually death. In rare cases, the rupture cicatrizes, and the patient recovers. Complete rupture usually causes death in a short time. The fœtus escapes partly or completely into the abdominal cavity. If the patient survives the immediate shock, fatal peritonitis soon ensues. In rare cases, the fœtus is shut in by adhesions, and the patient survives.

Perforations of the uterus may be produced by carcinoma, by abscesses in its neighborhood, and by ovarian cysts.

HÆMORRHAGE.

Effusion of blood into the cavity of the uterus occurs normally at every menstrual period. An excessive or deficient flow of blood at these periods is determined by causes which are described by the gynæcologists.

Effusions of blood at other than the menstrual periods may be caused by mechanical hyperæmia, by hæmorrhoids, by acute hyperæmia, by intra-uterine polypi, by acute and chronic inflammation, by typhus fever, scurvy, etc., by ulcerating carcinoma, by abortions and miscarriages.

A peculiar form of hæmorrhage is the polypoid hæmatoma, or fibrinous polypus of the uterus. It occurs after parturition and after abortions. The portion of the uterine wall where the placenta was attached, with or without a portion of retained placenta, forms the point of attachment of the pedicle of the polypus. We find a large, polypoid, bloody mass, firmly attached by a pedicle to the uterine wall. The uterus enlarges with the growth of the polypus; the cervix is dilated, and the thrombus projects into and may even fill up the vagina. The formation of such a thrombus is accompanied by repeated hæmorrhages.

Hæmorrhage in the substance of the uterus occurs in old age. The mucous membrane and uterine wall are infiltrated with blood, and there is some blood in the uterine cavity.

Retro-uterine, or *Peri-uterine Hæmatocele*, consists in an accumulation of blood around the uterus or in Douglas's cul de sac. It may consist of blood extravasated into the abdominal cavity, which settles into the pelvis; or, in consequence of local hyperæmia, there may be repeated extravasations of blood. In the latter case, the local peritonitis may produce false membranes, between the layers of which hæmorrhages take place. A similar condition more rarely occurs in the male.

The hæmorrhagic mass may be absorbed, or may soften, suppurate, and perforate into the rectum or vagina.

Some authors describe an extra-peritoneal hæmatocele which lies between the folds of the broad ligament. Tyler Smith believes that an extravasation may be inclosed by a secondary

false membrane. Most authors hold that the extravasation proceeds from the uterus, ovaries, and tubes. This is denied by Virchow, who holds that in most cases the blood comes from the vessels of membranes formed by local peritonitis.

The extravasation usually begins at a menstrual period, and increases at the succeeding periods.

INFLAMMATION.

1. *Of the Unimpregnated Uterus.*

Acute Catarrhal Endometritis is seldom seen after death. It occurs spontaneously, from gonorrhœa, with typhus fever, scurvy, etc. During life, the mucous membrane is swollen and congested, and covered with an increased quantity of muco-pus. After death, these appearances may remain or disappear.

Chronic Catarrhal Endometritis involves the body or cervix, or both. It may be the continuation of an acute inflammation, or begin as a chronic disease. It is found at every age; but is most common after puberty. It is produced by a great variety of causes. In the lesser grades of inflammation, we find no reliable lesions after death. In the more marked cases, the mucous membrane is thickened, grayish, or blackish colored, covered with muco-pus. The papillæ of the cervix are hypertrophied; the mucous follicles are swollen, and their outlets obstructed. The uterine wall becomes accid and atrophied, or may be hypertrophied, especially the cervical portion. This simple hypertrophy of the uterus from catarrhal endometritis is often mistaken for inflammatory enlargement of the organ. During the course of the inflammation, we find ulceration of the mucous membrane, especially in the cervix, and hypertrophy of the papillæ of the cervix. There may also result contraction or obliteration of the canal of the cervix. The inflammation may extend to the Fallopian tubes, or to the vagina.

Croupous Inflammation of the uterus is rare. It occurs with cholera, typhoid fever, and the exanthemata. The disease usually extends to the vagina, sometimes to the Fallopian tubes.

Acute Metritis is usually the result of acute catarrhal endome-

tritis. The organ is swollen, succulent, congested; the mucous membrane covered with muco-pus; the peritoneal coat congested. There may be small extravasations of blood in the wall or cavity of the uterus. The inflammation, in rare cases, becomes suppurative, and abscesses are formed in the uterine wall. These may perforate into the peritoneal cavity or into the rectum.

Chronic Metritis is the result of an acute metritis, or accompanies acute or chronic endometritis; or is idiopathic. The uterine walls become thick and hard. Many of the cases called chronic metritis are only catarrhal endometritis with hypertrophy of the uterus.

Perimetritis.—The peritoneal coat of the uterus may be inflamed with the production of membranous adhesions or of pus. The adhesions may be small or very extensive. The inflammation usually accompanies chronic metritis and endometritis. In prostitutes, adhesions are almost the rule.

Parametritis.—The connective tissue about the uterus, between that organ and the reflexions of the peritoneum, may be the seat of suppurative inflammation. It most frequently causes the death of the patient.

2. Of the Pregnant Uterus.

The same forms of inflammation which have just been described may attack the pregnant uterus.

Catarrhal endometritis may produce effusion of serum, extravasations of blood, and abortions.

Metritis may lead to softening of the uterine wall, so that ruptures take place during labor.

Perimetritis and parametritis produce adhesions and abscesses about the uterus.

PUERPERAL INFLAMMATION.

For a week after delivery, we find the inner surface of the still dilated uterus rough, especially at the insertion of the placenta, and covered with blackened, gangrenous-looking shreds of blood, mucous membrane, and placenta. This condition is not to be mistaken for inflammation.

In endometritis, the internal surface is covered with a thicker layer of gangrenous material, and there are patches of croupous exudation which may extend into the vagina. The uterine sinuses are filled with thrombi, for the most part broken down into a puriform fluid. There may be also a diffuse purulent infiltration of the uterine wall, or circumscribed abscesses. Very frequently there are also peritonitis and pyæmic infarctions of the viscera.

ULCERATION.

Phagedænic or Corroding Ulcer.—This rare form of ulceration usually occurs in old age, without assignable cause. It begins in the cervix and extends gradually until it may destroy the greater part of the uterus. It may even invade the bladder and rectum. The ulcer is of irregular form, its base is rough and blackish, its walls indurated.

Syphilitic Ulceration may attack the vaginal portion or interior of the cervix, or the internal surface of the body of the uterus. It has the usual forms of the hard and soft chancre.

Catarrhal Ulceration is most common in the cervix. It accompanies catarrhal endometritis. The ulcers are superficial erosions or formed from inflamed follicles.

DEGENERATION.

Waxy Degeneration has been observed by Virchow in one case. The uterus was hypertrophied and the muscle-cells were infiltrated with the waxy material.

NEW GROWTHS.

Myoma.—These tumors, often called fibrous tumors, are very common in the uterus. They occur after puberty, and usually in advanced life. They are usually multiple, and may reach an immense size. Their favorite situation is the body of the uterus, rarely the cervix. According to their position we may distinguish a subserous, submucous, and intra-parietal form.

The subserous myomata grow from the outer muscular lay-

ers of the uterus in the form of little nodules. As they increase in size, they separate themselves from the uterine wall and remain attached only by a narrow pedicle, or by a little connective tissue. If they are situated on the sides of the uterus, they may work their way between the folds of the broad ligament until they are at some distance from their point of origin. Some authors mention cases in which the tumors became entirely detached from the uterus and were free in the abdominal cavity.

In some cases, the tumors excite inflammation of the adjacent peritoneum, leading to the formation of adhesions or of collections of pus.

In other cases, the tumor reaches a large size, but remains firmly attached to the uterus. This organ is then drawn upward, the cervix and vagina are elongated and narrowed. The traction may be so great that the body of the uterus is entirely separated from the cervix. The bladder may also be drawn upward, producing incontinence of urine and cystitis.

This form of myoma is usually hard and dense, the fibrous tissue more abundant than the muscular. Calcification frequently takes place.

The submucous myomata grow from the inner muscular layers of the uterine wall. They commence as rounded nodules which lift up the mucous membrane. Their usual position is the fundus uteri, sometimes the anterior or posterior wall, rarely the lateral portions. In the cervix they are very seldom found.

As the tumors increase in size, they project into the uterine cavity. They then remain continuous with the uterine wall, over a large area, or are attached by a large or small pedicle. They are well supplied with vessels. The uterus dilates with the growth of the tumor, and its walls are also thickened. The tumor may reach such a size as to entirely fill the cavity of the dilated uterus and project through the cervix into the vagina.

The submucous myomata are usually single, although there may be at the same time subserous and intra-parietal tumors.

These myomata are usually soft, of whitish or reddish color,

of a lamellated or reticulated structure. The spaces between the lamellæ or in the reticulum may be so large that the growth has a cavernous or cystic character.

If the polypus is of large size and projects through the cervix, it may drag down the fundus of the uterus and produce inversion of that organ.

Hæmorrhages take place from the surface and in the substance of these myomata. Inflammation, suppuration, and partial gangrene also occur. The surface of the tumor may look like a ragged, gangrenous mass.

In some cases, the pedicle of the growth is destroyed and the tumor is spontaneously expelled.

The intra-parietal myomata grow in the substance of the uterine wall, but if they attain a large size, project beneath the serous or the mucous coat. They are found in every part of the uterus, but are most frequent in the posterior wall.

The shape of the uterus is altered in every conceivable way by the presence of these growths; its cavity is narrowed, dilated, or misshapen; it undergoes flexion and version in every direction. The tumors may grow outward, between the folds of the broad ligament. They may sink downward and become attached to the posterior wall of the vagina, looking as if they grew from it. They may project into the cavity of the uterus, fill it up, and project through the cervix, but have no pedicle like the submucous form. The uterus is dilated, its wall hypertrophied or atrophied.

The muscular fibres may undergo fatty degeneration, and the tumor diminish in size; cases of entire atrophy in this way are reported. Or, the muscular tissue atrophies, the connective tissue increases in amount and density, and the tumor becomes smaller and harder. Calcification may also take place, especially in the smaller myomata, and convert them into a stony mass.

Some of these tumors are soft. This takes place in several different ways. The interstitial connective tissue may be converted into mucous tissue, rich in cells and infiltrated with mucus. Or the connective tissue may undergo an œdematous softening, while the muscular fibres atrophy. We then find the

tumor filled with small cavities containing serum. Or the soft consistence may be simply due to the preponderance of muscular tissue.

Occasionally we find in a portion or in the whole of a tumor an abundance of dilated blood-vessels, which gives the part a true cavernous structure, like that of an erectile tumor. These tumors may change their size suddenly from the variable amount of blood which they contain.

A very important change, which is sometimes found in these tumors, is the development of cysts in their interior, (fibrocystic tumors.) This takes place in those tumors which grow outward beneath the peritoneal coat. We find one or more cavities, communicating with each other, with rough, trabeculated walls. The appearance is more that of cavities than of cysts. There may be a number of smaller cavities, or they may fuse to form one large one. The fluid contained in the cavities is like synovia, or is mixed with blood. These cystic myomata may reach an immense size, and fill the abdominal cavity. The diagnosis between them and ovarian cysts is very difficult, and they have frequently been the subjects of fatal operations.

The intra-parietal myomata may give rise to profuse hæmorrhages; they may suppurate and become gangrenous; the intra-uterine ones may undergo spontaneous expulsion; those which project beneath the peritoneum may produce inflammation of the neighboring tissues.

In the cervix uteri, myomata are rare. They may grow as polypi beneath the mucous coat; or produce enlargement of the anterior or posterior lips; or may grow outward into the abdominal cavity. I have seen a cystic myoma of large size growing from the posterior wall of the cervix and the upper part of the vagina, which was mistaken for an ovarian cyst.

In the wall of the vagina, myomata are still more rare. They usually grow outward, toward the rectum or bladder; very rarely project inward as polypi.

Fibroma Papillare, Papilloma, occurs in the form of (1) small, soft, polypoid growths from the mucous membrane of the uterus or cervix; (2) as syphilitic condylomata of the

cervix; (3) as large papillary growths from the cervix. The structure is that of papillæ, or branching tufts formed of a stroma of connective tissue and vessels, and covered with epithelium. The larger growths are closely related to epithelial cancer, and after a time exhibit a growth of epithelial cells which places them unmistakably in that class.

In some of the smaller polypi, the vessels may be so large and numerous as to give the growth a cavernous structure. In others, the follicles of the mucous membrane are present in large amount.

Angioma.—Virchow describes a single case of a small cavernous angioma in the wall of the uterus.

Cysts.—In the cervix uteri the mucous follicles are frequently dilated so as to form cysts filled with colloid matter and epithelium—ovula Nabothi. Sometimes there is an inflammatory growth of connective tissue around these cysts, so that they resemble the molluscum of the skin. In other cases, the cysts may project from the mucous membrane until they take the form of a polypus. The same changes are found less frequently in the mucous membrane of the body of the uterus.

In rare cases, serous and dermoid cysts are found in the wall of the uterus.

Tubercles.—We sometimes find the cavity of the uterus and of the Fallopian tubes lined with a yellow, cheesy mass. This is said to be produced by the degeneration of multitudes of gray, miliary tubercles scattered through the mucous membrane.

Syphilitic Gummata are occasionally found in the uterine wall in the form of whitish, rounded nodules.

Sarcoma occurs as a metaplasia in myomata of the uterus, forming myo-sarcomata. There is a growth of round, fusiform, and stellate cells, and of mucous tissue replacing the muscular and fibrous tissue. At the same time, large and small cysts may be formed in the tumor.

Sarcoma also occurs as a primary growth from the uterine mucous membrane. The tumors may grow from a large part of the internal surface of the uterus, or they may assume a polypoid shape. In the latter case, large portions have been removed at successive operations, followed by recurrence.

Carcinoma.—The most frequent form of cancer of the uterus is classed by different authors either as a true carcinoma, or as an epithelioma or canceroid. So that we find some saying that epithelioma, and others that carcinoma, is the more frequent, while both really mean the same growth. This apparent discrepancy arises partly from the different significations given to the words epithelial and true cancer, and partly from the peculiar anatomy of the growth.

The distinction between true and epithelial cancer is one which was never very sharply drawn, and now that the epithelial character of all cancers is so strongly insisted on, the two classes are being brought more and more nearly together.

The anatomy of this form of growth is partly that of the epithelial and partly that of the true cancers. There is a stroma composed of connective tissue fibres and round cells in variable proportions. This stroma may be abundant or scanty, but is usually well marked. In the stroma are cavities of round, oval, and tubular shape filled with cells. The cells resemble somewhat the epithelium of the vaginal portion of the cervix, but are more irregular in shape. They are large, polygonal, or rounded, with one or two large nuclei. In rare cases, they have the form of cylindrical epithelium. The cells are sometimes closely packed, and form concentric nests; in other cases, they merely fill the alveoli.

According to the manner in which this new growth invades the uterus, the gross appearances vary.

(1.) We find at first, in the vaginal portion of the cervix in the submucous connective tissue, either nodules or a general infiltration of a whitish new growth. The cervix then appears large and hard. Very soon the mucous membrane over the new growth degenerates and falls off, the superficial layers of the new growth undergo the same changes. After this, the formation of the new growth and its ulceration go on simultaneously, producing first an infiltration, and then destruction of the cervix, and often of a part of the body of the uterus. The growth frequently extends to the vagina, the bladder, and rectum with the same destructive character, so that we often find the cervix and upper part of the vagina destroyed, and in their

place a large cavity, with ragged, gangrenous, cancerous walls. Less frequently the pelvic bones are invaded in the same way. Metastatic tumors in the lymphatic glands and viscera are rare. I have seen small secondary nodules in the lungs, formed of large, flat cells closely packed in nests.

Not unfrequently the ureters are surrounded and compressed by the new growth, so that they become dilated. The dilatation may extend to the pelves and calices of the kidneys.

(2.) The development of the new growth is more rapid than its destruction. We find large, fungous, soft masses projecting into the vagina and the canal of the cervix.

(3.) The ulceration is more rapid than the new growth. We find larger or smaller ragged, ulcerating surfaces, of which the floor and walls are composed almost entirely of gangrenous new growth and uterine tissue. The amount of the new growth which is left may be so small as to render the diagnosis between carcinoma and phagedænic ulcer very difficult.

(4.) The new growth begins in the cervix, and extends uniformly over the internal surface of the cervix and of the body of the uterus. The entire uterus is converted into a large sac, of which the walls are infiltrated with the new growth, while the internal surface is ulcerating and gangrenous.

(5.) In rare cases, the growth begins in the upper part of the cervix, or in the body of the uterus, while the lower part of the cervix is not involved.

(6.) In rare cases, the entire wall of the uterus is infiltrated with the new growth, and the organ is much enlarged.

Papillary Epithelial Cancer (cauliflower growth) is an infrequent form of disease. It is said that simple papillomata may, after a time, exhibit an increased cell-growth, and epithelial nests in their stroma. But the real relationship between simple papilloma and papillary epithelioma of the cervix is still not at all understood. The growth springs from the vaginal portion of the cervix. It appears as a lobulated, fungous mass composed of numerous tufts. It may reach such a size as to fill the vagina. The minute structure is that of tufts covered with epithelium. In the stroma of the larger tufts, in the connective tissue at the base of the growth, and in the wall of the

cervix, beneath the growth, we find alveoli filled with epithelial cells.

True Carcinoma is not very common in the uterus. The gross appearances resemble those of the more ordinary epithelial form of the growth. The tumor usually begins in the cervix, sometimes in the body of the uterus. It forms fungous masses projecting into the vagina, or growth and ulceration go on at the same time, or ulceration goes on faster than growth.

Infection of the surrounding tissues is frequent; metastatic tumors often occur.

Colloid Carcinoma is rare. It may involve the cervix or body. It has the ordinary structure of a fibrous stroma forming regular alveoli, containing gelatinous matter and cells.

PARASITES.

Echinococci have been observed in the body and neck of the uterus; they may rupture into the peritoneal cavity or into the vagina.

THE OVARIES.

MALFORMATIONS.

Both ovaries may be absent, the other organs of generation being also absent or undeveloped. Or the ovaries may be only partially developed.

Absence or arrest of development of one ovary is sometimes met with in well-formed individuals. It is sometimes accompanied by a low position of the kidney on the same side.

The ovaries may pass into the inguinal canal or into the labia majora, and remain fixed there through life. Less frequently they are found in the crural canal and the foramen ovale.

CHANGES IN SIZE.

Atrophy is a regular senile change. The Graafian follicles disappear, and the organ shrivels into a small fibrous body.

Atrophy is also produced by ascites, by chronic inflammation, or as an idiopathic change.

True hypertrophy of the ovaries does not occur, but the stroma may be increased in amount.

CHANGES IN POSITION.

In adult life, the ovaries may descend as herniæ into the inguinal or crural canal, the foramen ovale, or the umbilicus.

The position of the ovaries in the abdomen may be changed by the pressure of tumors, the traction of false membranes, etc. Rokitsky describes a twisting of the ovary on its axis, with twisting of the Fallopian tube around the ovarian ligament. It usually occurs in enlarged ovaries, and by the compression of the veins, leads to congestion and inflammation of the tube, ovary, and peritoneum.

HÆMORRHAGE.

Every menstrual period is accompanied by the effusion of blood into a Graafian follicle. Normally, the amount of blood is small, becomes solid, is decolorized, and then gradually absorbed. Sometimes the effusion of blood is much greater; the follicle filled with blood is as large as a pigeon's egg. The blood may remain in the follicle and be absorbed, or it may rupture it and escape into the peritoneal cavity. Death may ensue from the hæmorrhage, or the blood may collect in Douglas's cul de sac, and be inclosed in false membranes. Hæmorrhages also occur in follicles, which have become cystic.

INFLAMMATION.

Inflammation of the ovaries occurs most frequently in the puerperal condition, either as part of a general peritonitis, or as a primary affection.

With puerperal peritonitis, both ovaries are usually inflamed; they are swollen, congested, soft, infiltrated with serum or pus, or gangrenous. The inflammation may attack principally the capsule, the stroma, or the follicles. Inflammation of the capsule results in adhesions and collections of pus, shut in by false membranes; of the stroma, in abscesses and fibrous induration; of the follicles, in their containing purulent serum. If the inflammation of the ovary is the primary lesion, it is usually confined to one organ. The stroma of the ovary is infiltrated with serum and pus, and may contain abscesses of large size.

In other cases, the ovary itself is but little changed, but is surrounded by a mass of fibrinous and purulent exudation. Such idiopathic forms of inflammation may terminate in recovery ; or the abscesses may perforate into the rectum and vagina ; or the ovary is left indurated and bound down by adhesions ; or the patient dies from the violence of the disease.

Idiopathic inflammation of the ovaries unconnected with the puerperal condition is rare. It is confined to one ovary. Either the follicles, stroma, or capsule may be involved. The inflamed follicles appear enlarged, their walls thickened ; they contain bloody or purulent serum. The stroma becomes infiltrated with serum or pus ; and later, we find abscesses or fibrous induration of the organ. The inflammation of the capsule leads to formation of membranous adhesions between the ovary, Fallopian tube, and adjoining organs.

NEW GROWTHS.

Cysts.—(1.) The Graafian follicles may be dilated so as to form cysts. This usually occurs only in one ovary ; sometimes in both. The follicles become enlarged, filled with serum, the ovum disappears, and we find cysts with thin walls, lined with pavement epithelium, and containing serum. Sometimes we find the cyst lined with cylindrical or even ciliated epithelium. There are usually a number of such cysts, all of small size. Sometimes, however, one cyst will attain a very large size. The fluid contained in it will be serous, viscid, purulent, or mixed with blood. There may be fibrous outgrowths from the interior of the cyst-wall. Such cysts usually are found after middle life, but may occur during youth, childhood, and even in the fœtus.

A variety of this form of cyst is formed by the dilatation of a follicle containing a corpus luteum. A thin layer of clot is then found lining the walls of the cyst. Such cysts may communicate with the Fallopian tubes.

(2.) *Composite Colloid Cysts.*—This is the most common form of ovarian cyst. Either one or both ovaries are affected. The cysts are found from the size of a walnut to that of the pregnant uterus. The gross appearance varies according to the size, number, contents, and stroma of the cysts.

The growth consists of a fibrous stroma, in which are tubular follicles lined with cylindrical epithelium, and containing colloid matter and rounded cells. The cysts are formed by the dilatation of these follicles. There may be a great number of follicles equally dilated, so as to form a number of cysts of moderate size; or only a few follicles are enormously dilated to form a large multilocular cyst, with but few compartments. The walls of the cysts may fuse together, and be absorbed so as to form one large cyst divided by incomplete septa. The stroma in which the follicles and cysts are imbedded may be largely developed or very scanty. The walls of the follicles and smaller cysts are lined with cylindrical epithelium. They contain colloid matter mixed with a variable number of rounded cells. Sometimes the cells contain globules of colloid matter. The walls of the larger cysts are composed of fibrous tissue. They may be thin and membranous; or we find developed on their internal surfaces an intra-cystic growth composed of a fibrous stroma and tubular follicles. These secondary follicles may also be filled with colloid matter, and form cysts. The intra-cystic growths may be so large as to fill up the original cysts. Sometimes the intra-cystic growth presents very little dilatation of its follicles, so that the entire tumor has more the character of a solid growth than of a cyst. The contents of the larger cysts are serum or colloid matter of variable consistence and color, and mixed with blood or pus. The cyst-walls may become inflamed and covered with fibrine or pus. The cysts may rupture into the abdominal cavity, or may be discharged through the intestine or through the abdominal wall.

The structure of these cysts is, therefore, that of a cystic adenoma. In some of these cysts, the formation of fibrous tufts, covered with epithelium, becomes the most prominent feature of the growth. Here, as in the cylindrical-celled epithelial cancers, we find the close relationship between follicles lined with epithelium and tufts covered with epithelium.

In many of the cysts in which the formation of follicles and their dilatation is well shown, the growth of tufts to a limited extent is also seen. But there are other cysts in which the

cyst-cavity is filled with a mass of tufts, looking like a cauliflower. Strictly speaking, such growth should be classed with the papillomata, but their relationship to the adenomata seems to be very close. They are often supposed by clinical observers to be cancerous, on account of their fungous appearance; but they are of a totally different character.

Dermoid Cysts.—These cysts are single or multilocular. They may be no larger than a walnut, or of very large size. They contain a thick, greasy material composed of epithelium, fat, and cholesterine. A portion of the internal surface of the cyst-wall may have the structure of the cutis: papillæ, hair follicles, and sebaceous follicles. In the cavity of the cysts may be found masses of hair, teeth, bones, cartilage, striated muscle, nerve-fibres and cells, and gray nerve-matter. Such cysts may be congenital. They often last for many years without much inconvenience; but they may form adhesions and perforate into the adjoining organs.

Fibroma.—A number of tumors have been seen in the ovaries by different observers, which, although resembling each other in their gross appearance, have a different anatomical structure. They occur usually in one ovary, sometimes in both. They are usually small, but may attain an immense size. They resemble the hard uterine myomata in their gross appearance. They occupy the entire ovary, or only part of it. They may contain cysts, or the follicles in the portion of ovary that is left may be dilated so as to form cysts. They are composed of fibrous tissue alone, or mixed with a little smooth muscle, or with fusiform and round cells. They belong, therefore, to the classes of Fibroma, Myo-fibroma, and Fibro-sarcoma.

Enchondroma of the ovary is rare. Cartilaginous tissue is found in some composite and dermoid cysts.

Syphilitic Gummata in the form of white nodules have been observed in a few cases.

Tubercles very rarely occur in the ovary. They are described as cheesy nodules of some size.

Sarcoma of the ovaries is not frequent. It may be primary or metastatic. The tumors are hard and dense, or softer and of loose texture. They are composed of fibrous tissue with fusi-

form and round cells. They usually involve both ovaries, and occur before middle life.

Carcinoma is found as a primary and metastatic growth, and as a continuous infection from the neighboring organs. Medullary carcinoma is the most frequent form, but scirrhus, melanotic, and colloid cancer are sometimes found. The new growth may be joined with the development of cysts.

THE FALLOPIAN TUBES.

MALFORMATIONS.

Absence of both tubes occurs with absence of the uterus. One tube may be absent, with arrested development of the corresponding side of the uterus. Both tubes may be imperfectly developed, either of their ends may be closed, they may be inserted into the uterus at an abnormal place, they may terminate in two or three abdominal ostia.

CHANGES IN SIZE.

Dilatation of the tubes, with increase of their length, is produced by the accumulation of catarrhal secretion, or by a constriction of some portion of the tube.

Narrowing of the tubes is produced by inflammation or by masses of mucus, blood, or pus. The abdominal end may be bound down and closed by adhesions, or the uterine end may be closed. In old age, the lumen of the tubes becomes very small or is obliterated.

Rupture of a dilated tube sometimes takes place.

HÆMORRHAGE.

Hæmorrhage in the tubes has been observed, in Typhoid Fever, in puerperal women, with retroversion of the uterus, with abortions, in measles, with menorrhagia, with hæmatometra, and with tubal pregnancy. The blood may escape into the peritoneal cavity and cause peritonitis.

INFLAMMATION.

Catarrhal Inflammation of the mucous membrane of the Fallopian tubes is of common occurrence. It is connected with endometritis, with the puerperal condition, or is an idiopathic affection. Usually both tubes are involved, the mucous membrane is swollen, and there is a production of mucus and pus in the tubes. The inflammation may subside without leaving any lesions, but more frequently results in peritoneal adhesions, obliteration of the tubes, dilatation, and abscesses.

Dilatation of the tubes is usually produced by obstruction or obliteration of their uterine extremities. The dilatation is generally moderate, converting the tube into a tortuous sacculated canal, containing mucus or serum mixed with pus or blood. More rarely, the dilatation is excessive and cysts are formed containing several pounds of fluid, and which may be mistaken for ovarian cysts. Dr. Peaslee has reported a case in which death ensued from hæmorrhage into the cavity of such a dilated tube.

In some cases, the fluid in the dilated tubes is entirely purulent, forming abscesses in the tubes. These abscesses may rupture into the peritoneal cavity, or the pus may escape into a cavity shut in by adhesions, or may perforate into the intestines.

Suppurative Inflammation of the tubes is found in connection with puerperal peritonitis and metritis. The tubes are swollen, intensely congested and pus exudes from their abdominal ends. It is often difficult to determine whether the peritonitis or salpingitis is the primary lesion.

NEW GROWTHS.

Cysts of small size are often found on the tubes and broad ligaments. They are formed by dilatation of the follicles of the foetal organs:—the Wolffian Body, and the accessory ovary.

Besides these, serous and colloid cysts, sometimes of large size, are found which seem to be true new growths. Kiwisch says that cysts are also found between the folds of peritoneum which accompany the round ligaments.

Fibroma occurs in the form of small nodules in the wall of the tubes. They may become calcified.

Lipoma is found between the folds of the broad ligament. The tumors are of small size.

Myoma of the wall of the Fallopian tubes is very rare and the tumors are of small size.

Carcinoma is found in the tubes only as a secondary growth continuous with the same growth in the adjoining viscera.

EXTRA-UTERINE PREGNANCY.

Tubal Pregnancy.—The impregnated ovum is in some way hindered from passing into the uterus, becomes fixed in the tube, and is there developed. The embryo and its membranes are developed until they reach such a size that the tube surrounding them ruptures. This may take place in the first month, or not until the sixth. The ovum may remain in the tube after the rupture; or may escape into the peritoneal cavity still enveloped in its membranes; or the membranes may be ruptured and left in the tube. The rupture is generally attended with fatal hæmorrhage. In some cases, death is caused by the rupture of a dilated vein, while the tube is still intact.

In rare cases, death does not take place, and the foetus is shut in by adhesions and false membranes. The embryo soon dies. In favorable cases, there is a slow absorption of the soft parts of the foetus, the bones are separated and left imbedded in a mass of fibrous tissue, fat, cholesterine, and pigment. Or the foetus retains its shape and becomes mummified, and may then be incrustated with the salts of lime.

In unfavorable cases, degeneration and gangrene of the foetus take place rapidly, with inflammation and suppuration of the surrounding tissues. There may be perforation and escape of the broken-down foetus through the rectum, vagina, bladder, or abdominal wall. The patient may die from peritonitis or exhaustion, or may recover after the escape of the foetus.

In some cases, the foetus may escape through a rupture of the tube into the space between the folds of the broad ligament.

Tubal-Abdominal Pregnancy is produced by the develop-

ment of the ovum in the fimbriated extremity of the Fallopian tube. Adhesions are formed, so that the foetus is partly in the end of the tube and partly in the abdomen.

Interstitial Pregnancy.—The ovum is arrested and developed in the portion of the tube which passes through the wall of the uterus.

Ovarian Pregnancy.—The ovum is developed in its Graafian follicle. The existence of this form of extra-uterine pregnancy is doubtful.

Abdominal Pregnancy.—The ovum after escaping from the ovary does not enter the Fallopian tube, but becomes fixed at some point near it. It is surrounded by thickened peritoneum and developed in that position.

In all forms of extra-uterine pregnancy, the uterus becomes enlarged, and a sort of decidua is formed on its internal surface.

THE PENIS.

MALFORMATIONS.

Entire absence of the penis is met with in connection with great defects of development of the rest of the body.

Absence of the penis with proper development of the other organs of generation is rare. The urethra then usually opens into the rectum.

An abnormally small penis is found, with absence or arrested development of the testicles.

Absence of the prepuce has been observed in a number of cases. Congenital phimosi is also not infrequent.

Hypospadia consists in an arrest of development of the penis and scrotum. In its highest degrees, the penis is one half to one inch long, the glans penis small and resembling a clitoris. On the lower side of the penis is a deep cleft, lined with mucous membrane. Into this cleft the urethra opens at the root of the penis. The scrotum remains separated into two halves, resembling labia majora. The testes descend into their proper position on each side, or remain in the abdomen. If the testicles continue to develop normally, the individual has the appear-

ance and capacities of a man; if their development is arrested, the individual is small and has a womanish appearance.

In lesser grades of the same malformation, the two halves of the scrotum are joined and the penis is larger, but a part of the urethra remains open as a cleft.

Uterus Masculinus.—This malformation consists in an abnormal change in the transition from the foetal condition of the parts to their fully developed form. In the male, normally, the greater part of Müller's canal disappears, and its lower end forms the vesicula prostatica. In this malformation, Müller's canal is changed, as it is in the female, into Fallopian tubes, uterus, and vagina, while at the same time the testes, epididymes, vesiculæ seminales, and spermatic cord are formed as usual. In the lesser degrees of this malformation, we find, in the place of the vesicula prostatica, a pear-shaped sac, as large as a pigeon's egg, with muscular walls and an epithelial lining. This sac may be incompletely divided into a uterus and vagina, and it opens into the urethra. In the higher grades, we find a well-formed vagina and uterus. The uterus may or may not have Fallopian tubes. The testicles are usually retained in the abdomen or inguinal canals, and are small. The spermatic ducts run on the sides of the uterus and open into the urethra or remain closed. The penis and scrotum appear as in hypospadias, or are well formed. The appearance of the individual varies with the development of the testicles.

A rare modification of this malformation consists in the existence of hypospadias; of a vagina and uterus; of a Fallopian tube and ovary on one side, and of a testicle and spermatic cord on the other side.

HYPERTROPHY. ATROPHY.

Hypertrophy of the penis is sometimes caused by venous congestion from heart-disease; by long-continued masturbation, the corpus cavernosum losing its contractility; and in rare cases, by hypertrophy of the stroma of the corpus cavernosum.

Atrophy is sometimes produced by chronic ulceration of the glans penis, and sometimes follows atrophy of the testicles.

INFLAMMATION.

Balanitis, inflammation of the prepuce, is usually produced by gonorrhœa, or by accumulations of smegma. The skin is red and swollen, and may ulcerate. Condylomata may be formed, and adhesions between the prepuce and glans. The glans may ulcerate and the prepuce may be much thickened. If the prepuce is long, there is an inflammatory phymosis, and the products of inflammation accumulate within the swollen prepuce. In some cases, the prepuce becomes gangrenous.

Paraphymosis is produced by the retraction of a narrow prepuce behind the glans, with consequent stricture, inflammation, and gangrene.

Inflammation of the Corpora Cavernosa is produced by gonorrhœa, injuries, fistulæ, typhus fever, measles, and occurs in connection with inflammation of the connective tissue of the pelvis. It results in fibrous induration of portions of the corpora cavernosa; in rare cases, in abscesses or diffuse purulent infiltration; sometimes in gangrene.

Syphilitic Ulcers frequently occur on the glans penis and prepuce.

The indurated chancre is formed either from an excoriation in which a pustule is formed, or from a little nodule. The pustule breaks and its walls are infiltrated with small, round cells. The nodule softens, breaks down, and forms an ulcer, of which the walls are infiltrated with cells in the same way.

Phagedænic ulcers occur, and may destroy a considerable part of the penis.

Herpes of the prepuce occurs in the form of small vesicles, which may later become ulcers.

Erysipelatous and furuncular inflammation sometimes involve the skin of the penis.

NEW GROWTHS.

Papilloma is found on the prepuce and glans penis. It occurs in the form of little warty growths, or of composite, cauliflower masses, even as large as a fist. In either case, the struc-

ture is the same—hypertrophied papillæ covered with epithelium.

Fibroma diffusum, or elephantiasis, occurs in the prepuce. It consists in a growth of fibrous tissue from the deep layers of the cutis, and forms tumors, which may reach an immense size.

Cysts, Lipoma, Fibroma, Angioma, have all been observed in the skin of the penis.

Fibrous Tissue is sometimes developed in the corpora cavernosa, and may become calcified.

Epithelioma is not infrequent in the prepuce and glans penis. It takes the form of nodules, which ulcerate, or of papillary outgrowths. The structure is that of epithelial cancer with large, flat cells. The growth may involve the entire skin of the penis, and invade the deeper tissues. The inguinal glands may be secondarily affected.

Eberth reports a case in which a cylindrical-celled epithelioma of the rectum was followed by secondary nodules in the liver and in the corpus cavernosum of the penis.

Carcinoma of the penis is not common. It begins in the glans penis or prepuce, in the form of nodules or of a diffuse infiltration. The growth goes on to involve the corpora cavernosa; the inguinal glands become affected, and metastatic tumors may be formed in the viscera.

THE SCROTUM.

The skin of the scrotum is subject to the various forms of inflammation, which may affect any part of the integument.

In very rare cases, tumors containing a considerable portion of the foetal skeleton have been found in the scrotum.

Dermoid Cysts containing hair, bone, cartilage, etc., sometimes occur.

Lipoma and Fibroma are sometimes found in the skin of the scrotum.

Epithelioma, Chimney Sweep's Cancer, occurs in the form of small nodules in the skin, which ulcerate, and from which little fungous growths arise.

Fibroma diffusum, elephantiasis of the scrotum, forms tumors

which reach an immense size. The growth consists in a development of connective tissue from the deep layers of the cutis, and is sometimes accompanied by dilatation of the lymphatic vessels.

THE TESTICLES.

MALFORMATIONS.

Absence of all the organs of generation is found in connection with other extensive malformations.

Absence of both testicles, either with or without absence of the epididymes, spermatic cord, and vesiculæ seminales, occurs in rare cases. The scrotum is only indicated, or may contain the epididymes. The penis is small, and the individuals are small and poorly developed.

Instead of being entirely wanting, the testes may be imperfectly developed. The individuals are weakly and effeminate.

Absence of one testicle, with healthy development of the other, is more frequent. The corresponding epididymis and cord may be absent or present.

The spermatic cords and vesiculæ seminales may be absent or imperfectly developed on one or both sides, while the testes are normal.

Either one or both testicles may remain permanently in their foetal position, or may not descend into the scrotum for several years after birth. Their descent may even be delayed until the thirtieth year of life.

This condition may depend on an arrest of development in the testes, or the gubernaculum testis; on adhesions produced by intra-uterine peritonitis; on narrowing of the inguinal canal; on narrowing or shortening of the vaginal process of the peritoneum; or on abnormal size or position of the testicle.

Usually the malformation is confined to one testicle, and then is more frequent on the left side.

The testicle is found in the abdomen close to the mouth of the inguinal canal; in the inguinal canal, just below the external ring; beneath the skin in the perineum; or in the crural canal with the femoral vessels.

Sometimes, while the testis is retained, the epididymis and spermatic cord descend into the scrotum.

The retained testis is usually not fully developed, or undergoes fatty or fibrous degeneration. It may become the seat of malignant new growths. Sometimes, however, although both testicles are retained, the individual is capable of procreation.

In rare cases, the position of the testis may be changed, so that the epididymis and cord are in front.

The existence of a supernumerary testis has been asserted in some cases, but is rather doubtful.

In cases of abnormally early development, we sometimes find the testes in a young child as large as they should be in an adult.

INFLAMMATION.

Inflammation of the testicles is caused by injuries, exposure to cold, inflammation of the urethra, syphilis, and occurs with parotitis. The testes, epididymis, or tunica albuginea may be principally involved. Usually only one testicle is inflamed; sometimes both. The inflammation may extend to the vas deferens.

The inflammation may be acute or chronic. Acute inflammation is more frequent in the epididymis and the tunica albuginea. The organ is congested and infiltrated with serum. From this condition, it may return to the normal, or small abscesses may form in the testis and epididymis, and destroy the greater part of the organ. In other cases, the epididymis remains swollen and indurated for a long time; in other cases, it becomes atrophied.

Chronic inflammation occurs as a sequel of acute inflammation, or as an original condition. It usually affects the testis, but may extend to the epididymis and spermatic cord. The organ is slowly enlarged. The tubes of the testis are enlarged and filled with degenerated epithelium; the interstitial fibrous tissue is increased in amount. The testis and epididymis are then found to be composed of a mass of fibrous tissue, imbedded in which are cheesy nodules—the remains of the degenerated tubules. In some cases, nothing but fibrous tissue is found; in

others, there is calcification. Sometimes abscesses are formed, and these may perforate through the scrotum, and be accompanied by a growth of granulation tissue which projects as a fungous mass.

Syphilitic inflammation affects principally the didymis. The tunica albuginea is thickened, and bands of fibrous tissue extend into the organ. In the new fibrous tissue, there may be gummy tumors.

HYDROCELE.

Hydrocele of the tunica vaginalis consists in an accumulation of serum in the cavity of this membrane. It is usually confined to one side. It is caused by acute or chronic inflammation of the tunica vaginalis, by varicocele, or by general dropsy. The serum is found in small or in large quantities; it is usually transparent, may contain cholesterine, or be purulent, or be mixed with blood. When spermatic cysts of the epididymis open into the cavity of the tunica vaginalis, the fluid contains spermatozoa. The tunica vaginalis remains unchanged, or is thickened, or contains plates of bone, or is covered with polypoid fibrous bodies, which fall off, and are found free in the cavity of the sac. There may be adhesions between the layers of the tunica vaginalis, and in this way the fluid becomes sacculated. The testis is pushed downward and backward; it remains unchanged, or is atrophied.

Hydrocele of the processus vaginalis consists in an accumulation of serum in the cavity of the vaginal process of the peritoneum, which remains open after the descent of the testicle. There are several different varieties.

(a.) The vaginal process is entirely open, and there is a free communication with the peritoneal cavity. The serum may originate in the cavity of the peritoneum, or of the vaginal process, and passes freely from one to the other.

(b.) The processus vaginalis is closed in the inguinal canal, while its lower portion is filled with serum.

(c.) The processus vaginalis is closed about the testis, and the visceral layer of the tunica vaginalis is formed. The serum ac-

cumulates in the upper part of the vaginal process, which communicates with the peritoneal cavity.

(d.) The vaginal process is closed in the inguinal canal and over the testis; the serum accumulates so as to form one or more sacs between these two points. Inguinal hernia may complicate this form of hydrocele.

Hydrocele of the spermatic cord consists in a general œdema of the connective tissue of the cord, or in the development of circumscribed cysts in this connective tissue.

A peculiar form of hydrocele is produced by the accumulation of serum in the sac of an inguinal hernia, from which the intestine has become retracted.

HÆMATOCELE.

Hæmatocele of the tunica vaginalis consists in an effusion of blood into the cavity of this sac. It may be produced by an injury, or may complicate a preëxisting hydrocele. The effused blood soon degenerates, and we find the sac filled with a brownish fluid, or a thick, grumous mass. The tunica vaginalis is usually thickened. The testis remains normal, or is atrophied.

Hæmatocele of the spermatic cord occurs in rare cases as a diffused infiltration of blood in the connective tissue of the cord. Or blood may be effused into a hydrocele of the cord.

NEW GROWTHS.

Fibroma occurs in the form of dendritic or polypoid growths of the visceral layer of the tunica vaginalis. The tumors are of small size; their pedicles may atrophy, and the little bodies become free. They are usually found in connection with hydrocele. Small fibrous tumors have also been observed on the spermatic cord. Sometimes the entire testis is replaced by a mass of fibrous tissue.

Lipoma occurs as a lobulated growth arising from the connective tissue of the spermatic cord, from the tunica albuginea of the testis, and from the tunica dartos of the scrotum. The tumors may reach a considerable size, are entirely composed of fat, or may be combined with myxoma or sarcoma.

Myxoma occurs in the testis as a soft gelatinous growth, which may break through the scrotum, and appear as a fungous mass.

Enchondroma occurs in the testis in the form of soft, hyaline tumors, which may attain a very large size. In some cases, we find great numbers of peculiar papillary cartilaginous out-growths partly contained in the enlarged lymphatic vessels of the tumor. Combinations of enchondroma with cysts, sarcoma, and carcinoma are common.

Tubercle usually begins in the epididymis, and may extend from there to the testis. More rarely, it begins in the testis. The growth begins in the form of small, gray granulations, which become cheesy, and fuse together into dry, yellow masses of considerable size. They are accompanied by a chronic inflammation of the surrounding tissues, and the cheesy mass may perforate the scrotum, and project as a fungous growth. The growth may also extend to the vas deferens, vesiculæ seminales, and prostate.

Cysts.—Single, simple cysts containing serum, and dermoid cysts containing epithelium, are sometimes found in the testis. Cysts containing spermatozoa are found in the epididymis; they are probably formed by dilatation of the follicles of the Wolffian Body. Cysts containing serum alone are formed from the same follicles. Small serous cysts are also formed in the connective tissue beneath the tunica vaginalis of the testis and epididymis.

Compound Cysts, Hydatid Tumor, Cysto-sarcoma, Cysto-fibroma, Fibro-cystic Tumor, Cysto-adenoma.—These names are given to a growth in the testis which is not of very frequent occurrence. The organ is converted into a mass of cysts containing serum, or colloid matter. The walls of the cysts are lined with pavement or cylindrical epithelium; their internal surfaces are smooth, or there are intra-cystic papillary growths. Sometimes the smaller cysts are filled with epithelium packed in concentric layers. The stroma is composed of fibrous tissue, with few or many connective tissue-cells; sometimes of mucous tissue; sometimes nodules of cartilage, and rarely striated muscle are found. Or there may be a combination with carcinoma.

In the only example of this form of tumor which I have seen, the cysts were lined with cylindrical epithelium; the stro-

ma was composed of fibrous tissue, in which were a few minute nodules of cartilage, and, in a few places, long, narrow, striated cells looking like embryonic striped muscle.

These tumors are usually considered benign; but Billroth mentions a case in which the removal of the testicle was followed by the growth of a retro-peritoneal carcinoma.

Sarcoma occurs both in the testis and epididymis. The tumors may attain a considerable size, are soft, with little stroma, or with a mucous basement substance; the cells mostly fusiform, belonging to the medullary and myxo-sarcomata. In some cases, we find cysts and canals lined with epithelium, and containing fluid, colloid matter, or epithelial contents. The canals and cysts are probably formed from the spermatic tubules.

Myoma.—A new growth of striped muscular fibres has been observed in four tumors of the testicle, in cases which are described as cystoid growths. The muscle was in the form of fusiform cells, with well-marked striation. I can add two other cases. In one, the tumor was removed by Dr. H. B. Sands, in May, 1868. It occurred in a middle-aged man, and was eighteen months in growing. The tumor weighed twenty-eight ounces, was confined to the epididymis, inclosed in the thickened albuginea, was soft and succulent, and contained many cysts. It consisted of connective tissue-fibres; of large numbers of long, fusiform cells; of long, fusiform, nucleated cells resembling embryonic striped muscle; of similar cells with marked striations; of large, well-shaped, striped muscular fibres, inclosed in their sarcolemma; of large, oblong, and round striated bodies looking like portions of muscular fibres. In the second case, the tumor was removed by Dr. Wood. It was a compound cystic tumor. In a few places, there were numbers of long, narrow, striated cells.

Carcinoma usually involves only one testicle. It is most frequently primary. It begins in the testis or the epididymis, rarely in the tunica vaginalis. The tumors are usually large, soft, succulent, often hæmorrhagic, or may be cystic. They are composed of a stroma, forming rounded spaces, in which are large, nucleated, rounded, or polygonal cells. The tunica albu-

ginea expands with the growth and incloses it, even when of large size. More rarely the stroma is abundant, and the tumor is hard and scirrhus. Metastatic tumors of the inguinal and lumbar glands often occur.

Syphilitic Tumors occur in connection with syphilitic orchitis. The albuginea is thickened, and bands of fibrous tissue extend into the organ. In this new fibrous tissue we find cheesy nodules, sometimes of considerable size. The epididymis is rarely involved.

THE PROSTATE.

HYPERTROPHY AND ATROPHY.

Hypertrophy of the prostate is a frequent senile change; it is general or partial.

In general hypertrophy, the entire organ is enlarged, and may reach the size of a man's fist. The enlargement is symmetrical, or is most marked in one half or in the so-called middle lobe. The organ is hard and dense, or soft, or alveolar, containing numerous small openings, from which a turbid fluid exudes. These different appearances depend upon the character of the hypertrophy. The muscular and fibrous tissue alone may be increased, or at the same time the glandular tissue, or the glandular tissue alone.

In partial hypertrophy, we find circumscribed nodules of muscular tissue, or of muscular and glandular tissue. They are usually situated at the periphery of the organ, and project into the bladder. They may become detached from the prostate, and are found as small, movable tumors beneath the mucous membrane of the bladder.

Both forms of hypertrophy frequently produce retention of urine and changes in the bladder.

Atrophy of the prostate is sometimes seen in connection with atrophy of the testicles, and with castration. Sometimes the ducts of the glandular portion are enlarged, or there may be fibrous degeneration of the organ.

INFLAMMATION.

Inflammation of the prostate is caused by gonorrhœa, by injuries, or, more rarely, is idiopathic. It may run an acute or chronic course. The gland may after a time return to its normal condition, or is gradually converted into a mass of fibrous tissue filled with abscesses. The abscesses may perforate into the bladder, urethra, vesiculæ seminales, rectum, or peritoneum. Or the inflammation may extend to the connective tissue of the scrotum, or beneath the pelvic peritoneum. The pus may become thickened and cheesy, or even calcified.

NEW GROWTHS.

Myoma has been already described under the head of partial hypertrophies of the prostate.

Tubercle is found in connection with tubercles in the testicles, vesiculæ seminales, and kidneys. Miliary granulations become aggregated in large numbers, and form, after degenerating, large cheesy masses.

Concretions are formed in the ducts of the glands. The smallest are rounded, colloid, lamellated bodies, which are stained red by iodine. The larger are hard, yellow or blackish, rounded or faceted, and contain phosphate or oxalate of lime.

Carcinoma of the prostate is not frequent. It is usually primary. It is soft and medullary, and may involve the surrounding soft parts.

THE MAMMA.

The mammæ are racemose glands, imbedded in connective tissue and fat. During childhood, they remain small and undeveloped. At puberty, the glands become larger and the acini are increased in size and number. The acini are filled with small, rounded, epithelial cells; the ducts are lined with cylindrical epithelium. During pregnancy, there is a further increase in the size and number of the acini, and at full term, the epithelium becomes filled with fat globules. The milk is

formed by an emulsion of these fat globules in serum. In old age, after the menses have ceased, the epithelium atrophies, the acini collapse, and finally we find nothing but a mass of fibrous tissue and fat, in which are imbedded the ducts of the gland.

In the male, the mammary gland remains undeveloped through life. It consists of a few ducts and acini imbedded in connective tissue.

MALFORMATIONS.

Absence of both mammæ is only found in connection with other marked malformations.

Absence of one mamma has been observed in a few cases, with and without defective development of the corresponding half of the thorax.

Absence of one or of both nipples is more common.

Arrest of development of the mammæ is found in connection with arrest of development of the organs of generation, and, to a less degree, alone.

Supernumerary mammæ and nipples have been observed in a number of cases. The glands may all secrete milk during lactation.

Too early development of the mammæ is sometimes found in young children in connection with abnormal development of the organs of generation.

INFLAMMATION.

The nipple, during lactation, is often the seat of cracks or fissures of the skin, sometimes complicated with inflammation.

Syphilitic ulcers may occur on the nipple in the form of primary chancres and secondary mucous patches.

Chronic eczema is found affecting the skin of the nipple.

Erysipelatous inflammation of the skin over the mamma may have its origin in the fissures of the nipples, or in unknown causes.

Acute inflammation of the mamma occurs during lactation. The connective tissue beneath the gland, between the gland

and the skin, and surrounding the gland, may be the seat of the inflammation. If the connective tissue-stroma of the gland is the seat of inflammation, usually only circumscribed nodules are affected. The inflammation may resolve, or goes on to suppuration. The abscesses usually open through the skin and soon cicatrize. Sometimes, however, the abscesses become chronic, with induration and formation of granulation tissue about them. Rarely, they perforate into the pleural cavity. In new-born children, we sometimes find a painful swelling and redness of one or both breasts, with a secretion of thin milk. This inflammation usually subsides in a few days, but may go on to suppuration.

At the time of puberty, a subacute form of mastitis may occur in both sexes. The gland is somewhat swollen, hard, and painful ; rarely, there is a small secretion of milk.

In women, after puberty, subacute mastitis sometimes occurs unconnected with lactation. There are circumscribed, indurated nodules in the gland, which may resolve without marked symptoms, or may slowly suppurate.

Chronic inflammation and the formation of cold abscesses are rare. They occur in married and unmarried women after puberty. We find circumscribed, hard nodules in the breast, which may remain unchanged for years. Or they may break down slowly into abscesses, containing a thin pus with cheesy fragments. Several such abscesses may communicate by sinuses. They may become surrounded by a thick wall of fibrous tissue.

HÆMORRHAGE.

In young women, who suffer from amenorrhœa and dysmenorrhœa, small hæmorrhages sometimes occur on and in the mamma; at the time of the menses.

Contusions of the breast may produce extravasations of blood in the connective tissue. The blood may be afterward absorbed, or may remain as a hard mass surrounded by dense connective tissue.

NEW GROWTHS.

General Hypertrophy of the mamma is a rare lesion. It may affect one or both breasts. It usually is found in young, unmarried women; sometimes, however, in advanced life. The glandular acini are increased in number and size. The interstitial connective tissue is also much increased.

Cysts are formed by dilatation of the ducts and acini of the mammary gland; as new growths in the connective tissue; and as an accompaniment of some forms of new growth.

Cysts containing milk are formed, during lactation, by the dilatation of the glandular acini and ducts. In the latter case, they may reach an enormous size, and contain several pounds of fluid. The milk may become mixed with serum, mucus, blood, colloid matter, and coagulated fat.

The acini may be dilated so as to form a great number of little cysts, containing serum or mucus. Minute examination shows the change beginning in the terminal acini as a simple dilatation. The epithelium is at first preserved. This condition is found in young women, with a too rapid development of the gland; in old women, with its atrophy.

Dilatation of the glandular ducts produces cysts of larger size. These cysts may be simple, or incompletely divided by septa, or there may be papillary ingrowths from their walls.

Cysts are formed in the stroma of the gland, independently of the glandular tissue. Their walls are lined with pavement epithelium, their contents are serous or colloid fluid. Their size varies from that of a pea to that of a man's fist. They are found in small and large numbers. There may be hæmorrhages into their cavities, inflammation and calcification of their walls, and papillary ingrowths.

Fibroma Diffusum.—This process may be classed as a new growth or as a chronic inflammation. There is an increase of the fibrous stroma throughout the gland, the glandular tissue remaining intact. Afterward, the new connective tissue shrinks and becomes harder, the glandular tissue is atrophied, and there may be cystic dilatation of the acini and ducts. The entire process is often accompanied by a great deal of pain,

and the atrophied condition is difficult to distinguish from atrophied carcinoma. Sometimes the growth of connective tissue, instead of being general, is confined to one or more circumscribed portions of the gland. We then find one or more hard nodules, which may become atrophied and indurated in the same way as the diffuse growth.

Fibroma Nodosum.—Small, circumscribed, fibrous tumors in the stroma of the gland are described, but appear to be rare.

Fibroma Papillare occurs in the form of intra-canalicular growths. Little papillary fibrous growths are formed on the walls of the milk-ducts. As the growths increase, the ducts become dilated, and we find a solid tumor divided up by clefts and fissures in every direction. The clefts are lined with pavement of cylindrical epithelium.

Lipoma usually occurs as a diffused increase of fat about the gland. In this way the mamma may appear to be enormously hypertrophied. More rarely, we find smaller, circumscribed lipomata in the stroma of the gland. Billroth describes a large lipoma, which grew behind the pectoralis muscle, and pushed the mamma downward and forward.

Enchondroma of the mamma is very rare. A single case is described by Astley Cooper, and another by J. Müller. A few other cases are described of enchondroma combined with carcinoma.

Adenoma.—A new growth of glandular tissue, resembling that of the mammary gland, sometimes occurs. Rarely, the glandular tissue forms the principal part of the new growth, which is then soft and succulent. More frequently, there is at the same time a growth of connective tissue. The tumor is then firm, but succulent, circumscribed, and movable, (Paget's Mammary Glandular Tumor.) The connective tissue between the new glandular follicles may be rich in cells, and pass gradually into the structure of a sarcoma. For this reason, Virchow regards the entire growth rather as a sarcoma with persistence of the normal glandular tissue, than as a new growth of glandular tissue. It sometimes happens that in a breast, which has been for some time the seat of a hard adenoma, a carcinoma is afterward developed as a separate tumor.

Cystoid Adenoma ; Cysto-Sarcoma ; Adenoid Sarcoma.—

These names are given to a group of tumors of the breast which have a very complex structure. They usually occur in middle-aged women ; grow at first slowly, afterward rapidly, and may reach an immense size. In some cases, there is no recurrence after extirpation ; in others, there are local recurrences, and even metastatic tumors. When these tumors are cut open, they look like a solid mass separated by clefts and fissures, and in which are cysts. The solid tissue appears as a uniform mass, or as a loose tissue, or as if made up of tufts. It is white and hard, or pinkish and soft, or grayish and colloid. The general arrangement is that of dilated ducts filled with intra-canalicular growths. The intra-canalicular growths are tufts composed of connective tissue fibres and cells, containing blood-vessels, and covered with epithelium. Or they may contain glandular follicles. The extra-canalicular part of the growth consists of connective tissue-fibres, and cells, and of glandular follicles. The follicles are in variable numbers ; they resemble those of the normal gland, or are larger, and lined with cylindrical epithelium. They are regarded as part of the normal gland, or as a new growth, by different authors. The connective tissue may be nearly pure fibrous tissue, or may contain so many cells as to merit the name of a sarcoma, or may be partly mucous tissue. In different tumors, therefore, or in different parts of the same tumor, we find a preponderance of fibrous, cellular, mucous, or glandular tissue. The prognosis seems to depend upon the relative amount of the connective tissue-cells, and the consequent more or less sarcomatous nature of the growth.

Myxoma occurs in the form of polypoid, pendulous tumors, covered with skin, and hanging down from the breast. It is also found as a more diffused growth in the stroma of the breast. The glandular tissue may atrophy and disappear in the new growth or the milk-ducts may be dilated, or there may be intra-canalicular growths of mucous tissue. In the latter case the tumors may resemble the adenoid sarcomata.

Sarcoma occurs in the mamma in the form of circumscribed nodules, or of larger growths replacing the entire breast. The

tumors may be composed of round, stellate, or fusiform cells, or of pigmented cells. The basement substance may be fibrous and abundant, or scanty, or may be mucous tissue. The glandular tissue may disappear, or the milk-ducts may be dilated, or there may be intra-canalicular sarcomatous growths. These tumors are more frequent in young than in old women.

We find, therefore, that there are fibrous, mucous, and sarcomatous tumors, in which the milk-ducts are dilated, and there are intra-canalicular growths. The gross appearance of these tumors may be the same, although their structure and characters are different.

Carcinoma.—Epithelial cancer, of the flat-celled form, is of rare occurrence in the mamma. It begins in the skin covering the gland, or in the nipple. Billroth describes a single case in which the glandular acini were enlarged and filled with hypertrophied epithelial cells.

Förster says that epithelial cancer with cylindrical cells is of not infrequent occurrence, and has the same course as ordinary cancer.

True Carcinoma usually occurs as scirrhus, medullary, or atrophic carcinoma, sometimes in the alveolar or colloid form. It is, generally speaking, a disease of advanced life. The tumor begins as a small, hard nodule, which increases slowly or quickly, and becomes fused with secondary nodules. If it grows rapidly, it may attain a considerable size, the skin is involved, and ulceration takes place. If the growth is slower, the tumor is smaller and harder, and ulceration may not take place for a long time. In some cases, the growth is very slow, there is great retraction of the new fibrous tissue, and the entire mamma is converted into a small, hard mass, which hardly looks like a tumor.

The tumors are made up of a connective tissue stroma and of epithelial cells. The stroma is composed of connective tissue-fibres and cells in varying proportion. It is usually well marked, sometimes forms almost the entire tumor, occasionally is very scanty. The epithelial cells are of moderate size and regular shapes, or large and malformed. They contain one or more nuclei. These cells are arranged in long, tubular follicles,

or in irregular spaces; or are found in large numbers, apparently from the fusion of several follicles. Whether the new growth of cells begins in the normal glandular follicles, in the connective tissue, or in the lymphatic vessels, is still an open question.

If the cells are numerous and the stroma scanty, the growth is called a medullary cancer; if the cells are few and the stroma abundant, a scirrhous cancer; if there are hardly any cells and a dense, hard, retracted stroma, an atrophic cancer.

All these forms are accompanied by secondary and metastatic growths. The axillary glands, the skin of the thorax, the liver, the bones, and nearly all the viscera may be the seats of metastatic tumors.

Alveolar or colloid carcinoma is rare. It occurs in the form of a circumscribed nodule, with the usual structure.

PARASITES.

Echinococci contained in hydatid cysts have been seen in a few cases in the mamma.

THE MALE MAMMA.

There may be an abnormal number of mammae.

In young children, the mammae may be swollen and inflamed as in young girls.

Cases are recorded in which adult males possessed large mammae, which secreted milk.

Hypertrophy of the breasts from an increase of connective tissue, or of fat, sometimes occurs.

Cysts from the dilatation of the glandular ducts are not very infrequent.

Cysto-Sarcoma, Sarcoma, and Carcinoma have been observed in a few cases.

THE BONES.

MALFORMATIONS.

(1.) Defective Formation exhibits itself in the absence and imperfect development of the bones, as in acephalous monsters, in microcephalia, in absence or smallness of the extremities.

(2.) Excessive Formation is seen in the abnormal size or abnormal number of the bones.

(3.) The formation of Fissures is seen in cleft palate, hemi-crania, spina bifida, etc.

(4.) The fusion of bones is seen in the extremities and cranium.

(5.) The ossification of the epiphyses may take place abnormally early or abnormally late.

CHANGES IN SIZE.

(1.) *Hypertrophy*.—The bones are enlarged in all their dimensions, and retain their normal texture. This condition may obtain in all the bones, as with giants; may be a congenital abnormality of a single bone, especially of the fingers and toes; may be an acquired condition, as in the bones of a hydrocephalic skull after the hydrocephalic process is at a stand-still. The bone may be increased in all its dimensions or simply elongated. Chronic inflammation of the bone or adjoining soft parts; release of a bone from physiological pressure, as in luxations; the effort to compensate for the destruction of an adjoining bone, may all produce hypertrophy.

(2.) *Hyperostosis*.—The bones retain their normal length or breadth, but are increased in thickness. The change may involve an entire bone, or only a circumscribed portion of it. In the latter case, the growth has more the character of a tumor.

The periosteum is thickened, the surface of the bone is rough, irregular, often covered with osteophytes of various forms. The walls of the bone are increased in thickness, and often in density; the medullary remains normal, or is narrowed, or even obliterated.

This change is most frequently found in the long bones and in the cranial bones. It is produced by chronic periostitis, by osteitis, rickets, and sometimes appears to be an idiopathic process.

(3.) *Sclerosis* consists in a change in the texture of the bone tissue by which it becomes denser and heavier. The process by which this is caused is an osteo-myelitis with the production of osteoid connective tissue, in which the salts of lime are afterward deposited. Most frequently there is at the same time a periostitis with production of new bone on the surface. Thus in the cranium the diploe may become filled with compact bone, while there is also an increase of bone beneath the periosteum, and in this way all the cranial bones may become enormously thickened.

The sclerosis may be idiopathic, or be caused by chronic inflammation of the joints or other parts near the affected bone, or be the termination of a rarefying osteitis.

(4.) *Osteoporosis*.—This change consists in an increase of the size of a bone with diminution of its density. The compact tissue of the walls of the bone is converted into a spongy, cancellous structure filled with red medulla. The bone may seem to consist of a system of concentric lamellæ, the medullary canal may be filled up with cancellous bone, or remain open; the change may affect most of a bone or only a circumscribed portion of it.

This condition is produced by inflammatory changes.

ATROPHY.

1. The atrophy may be due to defective development of the bones, and, therefore, occurs in young subjects.

(a.) It may be congenital, and affect all the bones, as in dwarfs, cretins, etc.

(b.) It may be the result of rickets, and then takes the form of a shortening of the long bones.

(c.) It is produced by an abnormally early ossification of the epiphyses and the interosseous cartilages. Thus diseases of the joints (in children) producing ankylosis and ossification of the epiphyses ; ossification of the sacro-iliac inter-articular cartilage ; ossification of the cranial sutures, etc.—all produce an arrest of development in the bones affected.

(d.) It may be produced by any cause which diminishes the nutrition of the bone, as paralysis of the muscles, or joint diseases, which render the limb useless.

2. The atrophy affects bones which are already fully developed, and occurs in adults.

We may distinguish two forms of atrophy : an eccentric and a concentric.

In the eccentric form, the bone retains its size and normal contour ; but the medullary cavity is enlarged, and the walls of the bone become thinner and thinner, until they may be not thicker than a sheet of paper. In the concentric form, the bones become smaller from the outside ; but in the long bones, only the thickness and not the length is diminished. In the eccentric form, the space which is left by the atrophying bone is usually filled up with fat.

Want of use and old age are the usual causes of atrophy of the bones.

A circumscribed atrophy of the bone is produced by any continuous pressure, usually by tumors. In this way, the wall of a bone may be perforated into its medullary cavity, and even through the opposite wall. The atrophy may be accompanied by sclerosis of the bone around the perforation.

FRACTURES, DISLOCATIONS, AND WOUNDS.

These conditions are so fully described in works on surgery and surgical pathology that it is needless to describe them here.

INFLAMMATION.

Periostitis.—We may distinguish five varieties of inflammation of the periosteum.

(1.) *Simple Periostitis*.—In this form, we find the periosteum thickened, succulent, congested, infiltrated with lymphoid cells, its fibres swollen. It is less adherent to the bone, and more adherent to the surrounding soft parts. The increase of cells takes place principally in the inner layers of the periosteum. This variety of inflammation may terminate in degeneration of the new elements, and the periosteum returns to its normal condition, or it may be only the first stage of one of the other varieties.

(2.) *Suppurative Periostitis* may begin as a simple or as a purulent inflammation. The pus is formed in the inner layers of the periosteum, and between it and the bone. The outer layers of the periosteum may resist for a long time the suppurative process. The accumulation of pus dissects up the membrane from the bone, and leaves the latter bare. The pus thus formed may remain in this position for a long time, may be absorbed, may become dry and cheesy, or may burst through the periosteum, and form abscesses in the soft parts. The bone is separated from its nutrient membrane. It may remain unchanged, but more frequently necroses, or inflammation of the bone itself, may be set up. Such a periostitis may run an acute or a chronic course. Sometimes suppurative periostitis takes on a very malignant character. Pus is formed not only beneath the periosteum, but in it, forming abscesses filled with foul pus. The periosteum breaks down into a gangrenous, stinking mass, and the same change may affect the neighboring soft parts. The medulla may take part in the process, and break down into a purulent, gangrenous mass. Hæmorrhages may complicate the process. The lymphatic glands are enlarged and swollen; abscesses form in different parts of the body, and the patient dies with the symptoms of septicæmia.

(3.) *Fibrous Periostitis* is a very slow, chronic inflammation, producing thickening of the periosteum. It is produced by necrosis, chronic arthritis, chronic ulcers of the leg, etc.

(4.) *Ossifying Periostitis* results in the formation of new bone from the inner layers of the periosteum. The bone thus formed is, at first, of very variable shape. The osteophytes, as they are called, form a thin, velvet-like, villous layer; or they are little spiculæ; or they form larger, rounded masses; or they are a thick, uniform layer, extending over a large part of a bone. They are at first very loosely connected with the bone. The new bone has at first a loose, spongy character. It is formed of thin plates of bone inclosing large cavities filled with medulla. Layers of compact bone tissue are formed from the medulla on the sides of the original plates, and thus the medullary cavities are gradually filled up with bone. The new bone may thus become as compact, or even denser, than normal bone. The hyperostoses and exostoses thus formed may remain indefinitely, or they may gradually become smaller and disappear.

(5.) *Syphilitic Periostitis*.—The syphilitic diathesis may give rise to simple, purulent, fibrous, and ossifying periostitis. In addition, we frequently find developed in the periosteum gummy tumors. These are rounded masses, sometimes gelatinous, sometimes firmer, composed of round and fusiform cells. They may disappear, may undergo cheesy degeneration, may be converted into fibrous tissue, or may suppurate. The bone beneath them may be unaltered or become diseased.

OSTEOMYELITIS.

In most of the inflammatory processes which affect the bones, the medulla has an important share; so that many conditions described as osteitis are really osteomyelitis. It is customary, however, to reserve the latter name for those cases in which a large part of the medulla is inflamed, and the bone tissue is little or not at all involved.

Using the word in this sense, we may distinguish an idiopathic and a traumatic osteomyelitis.

(1.) Idiopathic osteomyelitis generally occurs in persons under twenty years of age. It usually affects one of the long bones, more rarely several bones at once.

The disease begins with a diffuse hyperæmia and serous infil-

tration of a considerable portion of the medulla of one of the long bones. The inflammation begins in the shaft, and soon extends to the cancellous tissue of one or both of the epiphyses. The hyperæmia is succeeded by a purulent infiltration, which does not usually, however, soften or form abscesses. The periosteum generally becomes inflamed, and abscesses are formed beneath it, in it, and in the surrounding soft parts. This, combined with the osteomyelitis, often produces necrosis of small or large portions of the bone, or even of an entire bone. In other cases, the periosteum is thickened, and the shaft of the bone is thickened and sclerosed.

The purulent infiltration of the medulla, after a time, dilates the medullary cavity, and may finally break through the wall of the bone.

The joints in which the diseased bone terminates are almost always secondarily inflamed. There may be only a serous or a purulent exudation; or the osteomyelitis may extend to the articular end of the bone and the cartilage, and produce severe and destructive inflammation.

In young persons, the epiphyses very frequently become separated from the shaft by the destruction of the cartilage which binds them together.

In bad cases, the medulla becomes broken down and gangrenous; the joints are soon involved; large portions of the bone necrose; the periosteum and surrounding soft parts become gangrenous; the veins become filled with thrombi; and pyæmic infarctions are formed in different parts of the body.

(2.) Traumatic osteomyelitis is produced by contusions, fractures, wounds, and crushing of bones, and by amputations. Its character is the same as that of the spontaneous variety.

Osteitis.—The periosteum, the medulla, and the blood-vessels are essential component parts of a bone. They are the parts actively engaged in all inflammations of bone, while the bone-tissue proper, composed of cells imbedded in a calcareous basement substance, has but a passive share in the process. It is, therefore, more a matter of convenience than strictly proper to speak of periostitis, osteomyelitis, and osteitis, and the division is entirely arbitrary. Nevertheless, it is very convenient

to separate these three varieties according as the periosteum, the medulla, and the hard bone are the parts principally involved.

We may distinguish several different varieties of osteitis.

(1.) Rarefying osteitis occurs both as an idiopathic disease and in connection with caries, necrosis, fractures, etc.

In cancellous bone-tissue, the medulla becomes reddish, gelatinous, or like granulation tissue, and is thickly infiltrated with lymphoid cells. The bony septa become thinner, and disappear to a greater or less extent, so that in extreme cases, instead of cancellous bones, we find only a mass of granulation-tissue. When the disease occurs in the articular extremity of a bone, the granulating medulla may send little off-shoots through the articular cartilage. The cartilage will then be perforated with numerous little holes, which may afterward become fused together, and destructive inflammation of the joint follow.

The walls of the shafts of the long bones may by this form of inflammation be converted into a loose, spongy bone-tissue. If, at the same time, an ossifying periostitis goes on, the bone is thickened but spongy; or sometimes there are concentric layers of compact bone separated by cancellous bone. In these cases, there is a production of granulation-tissue in the medullary cavities and the Haversian canals; little projections of this granulation tissue pressing against the adjoining bone cause its absorption in small spaces or lacunæ; these lacunæ grow larger, fuse together, and the compact bone is thus filled with cavities, or entirely destroyed. Virchow supposed that the cavities were formed by fatty degeneration, cell-proliferation, or enlargement of the bone-cells. These cells may indeed be found somewhat changed, but the active processes are carried on by the medulla and the tissue in the Haversian canals. Volkmann states that there may also be a formation of new blood-vessels in the osseous tissue.

In the inflammation and granulation of the surfaces of wounded bone, the process is the same, and often leaves a circumscribed depression on the surface.

Rarefying osteitis may terminate in osteo-sclerosis, in supuration, and in caries.

(2.) Condensing osteitis, or osteo-sclerosis, is, properly speaking, an osteomyelitis. The medulla is replaced by red granulation-tissue, which becomes transformed into layers of compact bone on the inner surface of the preëxisting bone. The cancellous bone may thus be changed into compact bone, and the medullary canals of long bones may be completely filled up. There is very frequently at the same time an ossifying periostitis.

The process is sometimes idiopathic, more frequently excited by some chronic irritation of the neighboring bone, or soft parts, or is the termination of rarefying osteitis.

(3.) Carious osteitis is a variety which has been frequently called tuberculosis of the bones. It occurs principally in the cancellous ends of the bones, and in the bodies of the vertebræ. There is a chronic osteitis with sclerosis, or rarefaction, followed by cheesy degeneration of the inflamed medulla, or of pus, if any be formed. We then find dry, yellow, cheesy masses in the diploe. The process may terminate in necrosis, or in caries.

(4.) *Syphilitic osteitis*.—The syphilitic poison may excite one of the varieties of osteitis already mentioned, or it may produce the so-called gummy growths.

An osteitis gummosa begins as a periostitis, most frequently of the cranial bones. The periosteum is thickened and infiltrated with cells; the vessels which extend from the periosteum into the bone are surrounded with a new growth of small cells. At this stage, if we strip off the periosteum, we drag with it the vessels surrounded by the new cell-growth, leaving the bone beneath with numerous small perforations extending inward. As the disease goes on, the cell-growth around the vessels, and the consequent holes in the bone, become larger and larger, and fuse together, until a circumscribed portion of bone is replaced with cellular tissue. Then cheesy degeneration sets in, with absorption and production of connective tissue. In this way, we find in the bones dry, eroded depressions or perforations, containing more or less cheesy matter, while the skin over them is unchanged. In other cases, suppurative inflammation accompanies the gummy formation, and involves the skin. The pro-

cess may only involve a few small spots, or may spread over a large part of a bone. The portions of bone surrounded by the diseased portions may then necrose.

(5.) *Suppurative osteitis, abscess of bone.*—This process occurs usually in the ends of the long bones. It begins with a rarefying osteitis. The medulla goes on to actual suppuration, the bone-tissue is destroyed, and a circumscribed cavity is formed in the bone, filled with pus, and lined with granulation-tissue.

Less frequently, abscesses are formed in the shaft of a long bone, by a circumscribed suppuration of the medulla.

These abscesses usually occur in old people. They last for many years, have little tendency to perforation, may gradually enlarge, and be accompanied by an ossifying periostitis, so that the bone is expanded.

Very rarely acute suppurative osteitis, with rapid formation of an abscess, and perforation, has been observed.

CARIES.

The word caries is used by many as synonymous with chronic osteitis. It is convenient, however, to limit the term to one variety of osteitis—the ulcerative. In this sense, caries of bone corresponds to ulceration of the soft parts.

One of the simplest forms of caries is produced by chronic, suppurative arthritis. The articular cartilages are destroyed, the bone beneath undergoes molecular destruction, so that the cancellous diploe are laid open. The medulla then takes on an ulcerative inflammation.

In other cases, we find a rarefying osteitis, with a destructive, ulcerative character. At some portion of the inflamed bone, either on its surface or in its cancellous tissue, the inflamed medulla suppurates, and is destroyed with the corresponding portions of bone.

Sometimes this process is combined with cheesy degeneration of the products of inflammation, so that dry, yellow masses are found in the diploe, and replacing the destroyed bone.

The minute changes are very similar to those in chronic os-

teitis. The bone is destroyed both by the pressure of the granulating medulla and by a molecular destruction, forming irregular cavities, in which a sort of chalky dust can often be found. The basement substance of the bone may become granular, or separate into flakes, or give up its calcareous salts, and become fibro-cartilaginous. The bone-cells may be enlarged, fatty, or contain an increased number of nuclei.

NECROSIS.

By necrosis we understand the death of a larger or smaller portion of bone. This condition is induced by causes which deprive the bone of its proper vascular supply from the periosteum and medulla. Suppurative periostitis, osteomyelitis, and osteitis, traumatic separation of the periosteum, ulcers of neighboring soft parts, emboli, and diseases like typhus, which diminish the vitality, may cause necrosis.

When a portion of bone has died, an inflammation is set up at the dividing line between the dead and living bone. This inflammation has the characters of a rarefying osteitis, and finally separates the dead from the living bone. The dead bone, or sequestrum, may remain smooth and unaltered, or it may be eroded by the pressure of surrounding granulations.

Necrosis may involve the superficial layers, or the entire thickness of the wall of a long bone; or only the spongy tissue and inner layers of the wall; or an entire bone; or a number of different portions of the same bone.

The death and separation of the bone is very soon followed by the growth of new bone to repair the loss. The periosteum, the medulla, and the surrounding soft tissues may all take part in this new growth. The new bone is usually irregular, rough, perforated with openings, and incloses and takes the place of the sequestrum more or less completely.

RICKETS.

Rickets is a disease of the bones affecting their growth, which prevents the proper ossification of the growing bone.

The disease usually occurs during the first two years of life,

but may be congenital, or may occur as late as the twelfth year.

The physiological growth of bones depends upon three conditions. They grow in length by the production of bone between the epiphysis and diaphysis; in thickness, by the growth of bone from the inner layers of the periosteum. At the same time, the medullary canal is enlarged in proportion to the growth of the bone by the disappearance of the inner layers of bone.

In rickets, these three conditions are abnormally affected. The cell-growth, which precedes ossification, goes on with increased rapidity and exuberance both between the epiphyses and diaphyses, and beneath the periosteum, while the actual ossification is imperfect or entirely wanting. At the same time, the dilatation of the medullary cavity goes on irregularly, and to an excessive degree.

The increased cell-growth between the epiphyses and diaphyses produces the peculiar knobby swellings at the ends of the bones which are characteristic of rickets. At the same time, the medullary cavity increases rapidly in size, the inner layers of the wall of the bone become spongy, the medulla may be congested, its fat disappears, fusiform cells and connective tissue are developed along the course of the muscles, and the process may take on the character of an osteitis.

The result of these processes is, that the bones lose their solidity, and can no longer resist the traction of the muscles or outside pressure. The epiphyses may be displaced or bent, especially in the ribs, less frequently in the long bones. The long bones and the pelvic bones may be bent in a variety of forms. Incomplete fractures are not infrequent; complete fractures do not usually occur until the later stages of the disease, when the bones become solid again. In the head, the cranium is unnaturally large for the size of the face. The fontanelles and sutures remain open, the bones are soft, porous, hyperæmic, and, at their edges, there are rough bony growths beneath the pericranium. Sometimes, especially in the occipital bone, there are rounded defects in the bone, filled only with a fibrous membrane.

After a time, the rachitic process may stop, and the bones take on a more normal character. The porous bone tissue becomes compact and even unnaturally dense, the swellings at the epiphyses disappear, many of the deformed bones may become of a normal shape. In severe cases, however, the deformities continue through life; especially is there a cessation of the growth of the bones in their long axis, so that the persons affected are dwarfed.

The disease may have an acute or a chronic character.

The acute form begins usually during the first six months of life. The children suffer from vomiting, diarrhoea, profuse sweating, chronic bronchitis and pneumonia, general anæmia, and wasting. They either die, or the rachitic process is gradually developed.

The chronic form is seen in older children, and often in those apparently healthy. The changes in the bones may take place without any constitutional symptoms, though there is often catarrhal bronchitis, pneumonia, and anæmia.

OSTEOMALACIA.

This disease consists in a softening of the adult bones. It occurs in adults, most frequently in females during pregnancy and after parturition; rarely in adult males and females without known cause. In puerperal women, the disease begins in the pelvic bones; in other cases, in the vertebræ and walls of the thorax. In all cases, it has a tendency to involve a large part of the entire skeleton.

The disease begins with a simple disappearance of the calcareous matter around the Haversian canals and medullary cavities, leaving the bone-cartilage and bone-cells intact. Afterward this osteoid tissue breaks down, and is replaced by the exuberant medulla.

When the disease is fully developed, we find the medulla intensely congested, the fat-cells disappear, the lymphoid cells are increased enormously in number. The congestion may then diminish, and the medulla looks like a pale jelly. The compact bone becomes soft and spongy, the walls of the diploe become thinner and thinner until, in the worst cases, there is

nothing left but a layer of periosteum and thin cartilage, filled with a soft mass of medulla, connective tissue, blood-vessels, and serum.

In consequence of these changes, the bones become bent, completely and incompletely fractured, contracted and atrophied.

NEW GROWTHS.

Osteoma.—The bony tumors which grow from the bones are called exostoses or enostoses, according to their origin from the surface or interior of the bone.

They may contain all the constituents of a normal bone: bone, medulla, vessels, periosteum, and cartilage. The new bone may be compact and like ivory, or spongy, or contain large cavities filled with medulla. The shape of the exostoses varies very much. They may be in the form of sharp, narrow spiculæ and processes; they may have a polypoid shape, with a narrow pedicle; they may form rounded tumors, with a broad base; or they may have more the character of an hyperostosis.

The bone beneath them may be normal, or sclerosed, or rarefied, or the medullary cavity of the bone may communicate with that of the exostosis.

Exostoses are usually developed from the periosteum, sometimes in the insertions of the tendons and muscles.

A peculiar form is that called exostosis cartilaginea. It consists of a bony tumor covered with a thin layer of cartilage. Sometimes these growths are covered with a synovial sac, which may communicate with the cavity of a joint. They have a tendency to enlarge, and grow, like the long bones, from the inner layer of the cartilage which covers them.

Enostoses are developed in the interior of bone from the medulla. They may increase in size until they project like an exostosis. Their most frequent situation is the bones of the cranium and face.

Exostoses may be single or multiple, and may occur at all ages, even during intra-uterine life.

Enchondroma.—Tumors composed of cartilage usually grow from the interior of the bones.

They are composed of cartilage with a hyaline, mucous, or fibrous basement substance, or with all these varieties combined in the same tumor. They are usually divided into lobules by bands of fibrous tissue, and this fibrous tissue may form a large part of the tumor. They may calcify, ossify, and exhibit a great variety of transitions and combinations, with osteoma, myxoma, lipoma, sarcoma, and carcinoma.

They occur most frequently in the small bones of the hand and foot, in diminishing frequency in the tibia, femur, and humerus, lower jaw, pelvic bones, scapula, ribs, upper jaw, and fibula.

They are most common in young persons, and may be congenital. They may be single or multiple.

Their prognosis depends upon their exact anatomy. If they are purely cartilaginous, metastases are rare, but local recurrences sometimes happen. If they are combined with myxoma, sarcoma, or carcinoma, the prognosis becomes worse, and may be very bad indeed.

Under the name of *ecchondroma*, Virchow describes a form of cartilaginous tumor which grows from permanent cartilage. The tumors are usually small, and of little clinical importance. They grow from the cartilages of the ribs, the pelvic synchondroses, the intervertebral cartilages, the cartilages of the larynx and trachea, and the speno-occipital synchondrosis.

Fibroma.—Fibrous tumors may grow either from the periosteum or the medulla. The ordinary seat of the periosteæ fibromata is the bones of the head and face. They form polypoid tumors, projecting into the posterior nares, pharynx, mouth, and the antrum of Highmore. On the other bones they are not common.

The central fibromata, growing from the medulla, are rare. They usually occur in the lower jaw, but have been seen in the ends of the long bones, the phalanges of the fingers, and the vertebræ.

The fibromata may calcify, ossify, contain cysts, and be combined with sarcoma and angioma.

Angioma—Aneurism of Bone.—A very large number of the tumors, which have been described under these names, are

really sarcomata, or other tumors which happened to be very vascular. Some authors, indeed, are disposed to deny altogether the existence of real vascular tumors in bones. There are, however, reliable cases of cavernous angiomata growing between the periosteum and bone, and intimately connected with the latter. Whether myelogenic angiomata occur is doubtful.

There are several cases described of cavities filled with blood in the interior of bones, which it is difficult to interpret. They have mostly been found in the head of the tibia. They are said to have consisted of single sacs composed of thickened periosteum lined with plates of bone, and filled with fluid and clotted blood. No large vessels communicated with the sacs, but their walls were covered with a rich vascular plexus, branches of which opened into the cavity of the sac.

Cystoma.—The cysts which occur in bones are new growths, not retention cysts. They are not to be confounded with cystic degeneration of tumors of the bones. They are found, almost exclusively, in the maxillary bones. They are usually monolocular, contain clear serum, or a mucous, or viscous, or brown fluid, sometimes containing cholesterine. The cyst-wall is a fibrous sac lined with epithelium. They begin in the interior of the bone, and, as they increase in size, expand it until they are only covered with a thin shell of bone. They may reach a large size, even as large as a child's head.

Tubercles are of rare occurrence in bone. The products of inflammation not infrequently undergo cheesy degeneration and become crude, yellow masses, which are often called tubercular, but are not so. True tubercles occur in the medulla, especially in the bodies of the vertebræ, and the ends of the long bones. They are at first small, miliary, grayish nodules, disseminated in large numbers. They afterward become cheesy, and are surrounded by a zone of suppurative osteo-myelitis. In this way caries and cavities in the bone are formed.

Sarcoma.—This form of tumor is especially common in the bones. It grows from the inner layers of the periosteum, or from the medulla, so that we may distinguish a periosteal and a myelogenic sarcoma. Sometimes the tumor attacks the bone

itself so early that it is impossible to say whether the tumor began in the periosteum or in the medulla. There is also a variety which grows close to the outside of the periosteum, and becomes connected with it—parosteal sarcoma.

The periosteal sarcomata usually belong to the varieties fibro, myxo, chondro, and osteo-sarcoma, more rarely to the medullary variety. They are very frequently combined with fibroma, chondroma, and osteoma. They commence from the inner layers of the periosteum, pushing this membrane outward. After a time the periosteum is attacked, and the tumor invades the surrounding soft parts. The bone beneath may remain normal, or may be eroded and gradually disappear until the tumor is continuous with the medulla. Portions of the tumor may be calcified, or a growth of new bone may accompany its growth. The new bone usually takes the form of plates, or spiculæ, radiating outward. The minute anatomy of these tumors is very variable. The simplest—the fibro-sarcomata—are composed of fusiform, round, stellate, and sometimes giant, cells, (myeloplaxes,) in variable proportions, packed closely in a fibrous stroma. In the medullary form, the stroma is diminished to a minimum, and the round cells are most numerous. In the chondro and myxo-sarcoma, the basement substance may be hyaline, or mucous, and the cells follow the type of cartilage and mucous tissue. In the osteo-sarcoma, there is a new growth of osteoid tissue, more or less calcified. In the chondro, myxo, and osteoid sarcomata, there are usually fusiform, round, stellate, and giant cells, in addition to the cartilage and bone-cells.

The myelogenic sarcomata commence in the medulla and grow rapidly. The bone surrounding them is destroyed, and they project as rounded tumors. Most frequently, new bone is formed beneath the periosteum, so that the tumor is inclosed in a thin, bony shell; sometimes there are also plates of bone in the tumor; sometimes the periosteum is unaltered; sometimes it is perforated, and the tumor invades the surrounding soft parts. The tumors are usually very soft, vascular, and hæmorrhagic, or may contain cysts. They are sometimes mistaken for cysts filled with blood. They are composed principally of

round cells, sometimes of fusiform cells, and frequently contain great numbers of myeloplaxes.

The parosteal sarcomata resemble the periosteal. They appear to grow from the outer layers of the periosteum or from the adjoining connective tissue. They may be as firmly connected with the bone as the periosteal form. The periosteum may remain intact between the tumor and the bone, or it may disappear and leave them in apposition.

Osteoid Chondroma.—This form of tumor is found usually at the ends of the long bones. The tumors grow between the periosteum and the bone, forming sometimes enormous growths. They have a pyriform shape, are not lobulated, are of a smooth, hard consistence, with some calcified portions. Their minute structure is that of bone cartilage, osteoid tissue, a hard, homogeneous basement substance, in which are imbedded round, fusiform, and stellate cells. Blood-vessels ramify through the tissue, and may be arranged as if in Haversian canals. There is only needed a deposit of the salts of lime to convert this tissue into bone, and this change may take place over a large portion of the tumor.

Carcinoma.—The subject of true cancer of the bones is, in many respects, very obscure. Of the older tumors described under this name, a very large number are sarcomata. This is especially the case with primary tumors. Secondary and metastatic tumors can be more definitely made out. They follow the anatomy of the primary tumors, and may be scirrhus, medullary, or epithelial.

Epithelial cancer of the lips and uterus not infrequently directly invades the adjoining bones.

A peculiar form is the development of metastatic nodules in the bones. The primary disease is usually an atrophic carcinoma of the mamma, and the nodules may be found in nearly every bone in the body. They grow in the medulla in the form of hard, white nodules. The bone near them may simply disappear so that fractures occur, or the bone may lose its salts of lime and become soft. In this way, marked curvatures of the long bones may take place.

PARASITES.

Echinococci are sometimes found in bone. They have the usual form of a mother cyst with daughter cysts. They grow in the bone tissue, but the cysts may burst beneath the periosteum or in the medulla.

Cysticercus cellulosæ has been seen in a single case in the first phalanx of the middle finger.

THE JOINTS.

MALFORMATIONS.

One or more of the joints may be absent, or may be in excess, corresponding to the absence or excess of the bones forming them. Some of the ligaments may be absent. There may be congenital luxations, fractures, and synostoses.

CHANGES IN POSITION.

Luxations may be complete or incomplete. They may be intra-uterine, or produced by violence, or by relaxation of the ligaments, or by chronic inflammation of the joints. A full account of dislocations will be found in works on surgery.

INFLAMMATION.

Acute Synovitis.—The earlier stages of acute inflammation of the synovial membranes are better known from experiments on animals than from post-mortem examinations. The first changes are swelling and congestion of the membrane, with increased growth and desquamation of epithelium and infiltration of the membrane with lymphoid cells. These conditions are soon followed by an exudation.

The exudation may be a clear serum, in which epithelial cells, lymphoid cells, and sometimes blood, will be found. Or floculi of fibrine may float in the serum, or the fibrine may be in excess and the serum nearly absent. Or there is an excessive production of lymphoid cells, and the synovial sac is filled with pus.

Serous Synovitis may be caused by contusions, penetrating

wounds, gonorrhœa, rheumatism, or be idiopathic. The distension of the sac with serum is the most prominent lesion. The disease terminates in recovery or becomes chronic.

Croupous Synovitis leads to adhesions and ankylosis of the affected joint.

Purulent Synovitis may terminate in recovery with a normal joint. Or the articular cartilages ulcerate and are destroyed, the exposed bone may become carious, the pus may break through the capsule of the joint and form large abscesses in the soft parts.

Sometimes the inflammation is not only suppurative, but gangrenous, and runs a rapidly fatal course. The synovial membrane, articular cartilages and ends of the bones all undergo a rapid suppuration and gangrene.

Pyæmia and septicæmia, small-pox, measles, scarlet fever, diphtheria, mumps, typhus fever, glanders, the puerperal condition, exposure to cold, penetrating wounds, and injuries may all give rise to purulent synovitis.

Chronic Synovitis begins as such, or is the result of a previous acute inflammation.

There is an increase of fluid in the joint. This fluid is thin and serous, or is thickened with flocculi of fibrine and epithelial and lymphoid cells, or is thick, syrupy, or even gelatinous.

The synovial membrane is at first congested, its tufts prominent. Later it becomes thickened, sclerosed, and anæmic, the epithelium is destroyed, and the tufts become large and projecting. From the distension of the capsule there may be subluxations or luxations of the joint, or the capsule may be ruptured. Recovery is rare.

Chronic Rheumatic Arthritis is a chronic inflammation of the joints, most common in elderly persons. Usually several joints are affected and disease, advances slowly and steadily. There is a chronic thickening of the synovial membrane and the fibrous tissue adjacent to it. The articular cartilages degenerate and disappear. The contracting synovial membranes and fibrous tissue render the joints stiff and deformed. Not infrequently fibrous and bony ankyloses are formed between the ends of the bones.

Chronic fungous and purulent Arthritis. Tumor albus. Arthrocase. Strumous Arthritis.

These names are used to designate a chronic inflammation which may involve all the parts of a joint. The larger joints are the ones usually diseased, and children and young persons the most disposed to the disease. The so-called scrofulous diathesis is said to predispose to this inflammation, but local injuries are frequently the exciting cause.

The disease begins usually in the synovial membrane, sometimes in the ends of the bones.

The synovial membrane is thickened, congested, loses its epithelium and is converted into granulation tissue. This new granulation tissue appears red and succulent, or hard and yellow from degenerative changes, or œdematous. A variable amount of pus in the joint is produced at the same time with the changes in the synovial membrane, and may rupture the capsule.

These changes are accompanied by a thickening of the fibrous tissue around the joint. We then find around the joint a mass of hard, dense, sclerosed, anæmic tissue, composed principally of fusiform cells. In other cases, the new tissue is soft, succulent, vascular, composed of round cells.

This new tissue may become so large as to replace all the soft parts above it, and become continuous with the skin around the joint. In some cases, portions of the new tissue break down into pus and form periarticular abscesses.

The articular cartilages become partially or entirely destroyed. The cartilage cells increase in number, diminish in size so as to resemble lymphoid cells, and degenerate, the cavities in which they are contained enlarge, the basement substance of the cartilage undergoes a kind of mucous softening. These changes begin on the surface of the cartilage and work inward until they have destroyed a smaller or larger part of it.

If the disease begins as an osteitis of the end of the bone, instead of as a synovitis, the medulla of the bone becomes converted into granulation tissue, which grows into the cartilage and perforates into the joint by a number of sieve-like openings.

The ends of the bones are found in a condition of caries, necrosis, sclerosis, fatty atrophy, or overlaid with osteophytes.

The disease always runs a very chronic course and may destroy the patient's life. If recovery takes place before the cartilages and bones are involved, the joint is preserved; but is stiffened, or even immovable, from the contraction of the new fibrous tissue around it. If the cartilages and bones are diseased, the joint is destroyed, and either bony or fibrous ankylosis results. Sometimes from the change in the articulating surfaces, and the contraction of the muscles and the new fibrous tissue, partial or complete dislocations are produced.

Arthritis Deformans, Malum Senile Articulorum, Chronic Rheumatic Arthritis, Rheumatic Gout.—These names have been given to a species of chronic inflammation of the joints, producing, not destruction, but deformity. It occurs in elderly persons, and attacks one or several joints. It is idiopathic, or due to rheumatism, to injuries, or follows an acute arthritis.

The capsules of the affected joints are thickened and sclerosed. The synovial fluid is at first increased in quantity; later, diminished and thickened. The tufts of the synovial membrane become much enlarged and vascular; they may be converted into cartilage. Sometimes the capsule becomes ossified. The new bone grows from the edge of the cartilage within the capsule. Its articular surface is covered with cartilage.

The articular cartilages are much changed. The basement substance splits into tufts, while the cartilage-cells are increased in number. Or the basement substance becomes fibrous; or it is split into lamellæ and the cartilage-cells multiplied; or there is fatty degeneration and atrophy. As a result of these changes, larger or smaller portions of the cartilage are destroyed, and the bone beneath is laid bare. The exposed bone becomes compact and of an ivory smoothness.

The ends of the bones are much deformed. They are flattened and made broader by irregular new growths of bone, while at the same time they atrophy.

The new growth of bone starts from the articular cartilages. The cartilage-cells increase in number, and the basement substance in quantity. This growth is most excessive at the edge

of the cartilage, so that a projecting rim is formed there. This projecting rim then begins to ossify next the bone, and, at the same time, new cartilage is formed on its surface, so that we find large masses of bone covered with cartilage.

The atrophy of the ends of the bone, which goes on at the same time, is not so easily explained. It seems to be a kind of inflammatory atrophy taking place beneath the cartilage.

NEW GROWTHS.

Lipoma.—A new growth of fatty tissue may begin in the outer portions of the synovial membrane, push this inward, and project into the joint in a mass of tufts—*lipoma arborescens*.

Fibroma occurs as an hypertrophy of the little tufts and fringes of the synovial membrane. In this way, large polypoid and denticritic bodies are formed. The pedicles of these growths may atrophy and even disappear, so that the growths are left free in the cavity of the joints.

Corpora Aliena Articulorum, Loose Cartilages in the Joints. These names are given to bodies of various structure and origin, which are found free, or attached by slender pedicles in the cavities of the joints. They are most frequently found in the knee; next in order of frequency, in the elbow, hips, ankle, shoulder, and maxillary joints. They may be single, or in hundreds. Their size varies from that of a pin's head to that of the patella. They are polypoid, rounded, egg-shaped, or almond-shaped; their surface is smooth or faceted, or rough and mulberry-like. They are composed of fibrous tissue, cartilage, and bone in various proportions.

These bodies are formed in four different ways.

(1.) By hypertrophy of, and production of cartilage and bone in the physiological synovial tufts.

(2.) More frequently by a change into cartilage of portions of the synovial membrane. Small, flat plates of cartilage form on the inner surface of the synovial membrane. These plates increase in size and their outer layers ossify. They may remain fixed in the synovial membrane, or they project and become

detached from it. They then appear as flattened, concave bodies, composed of bone covered with cartilage on one side.

(3.) The growth of cartilage and bone begins in the outer layers of the synovial membrane, or in the periosteum, near the joint. The new growth pushes the synovial membrane inward, and projects into the joint as a polypoid body covered with the inner layers of the synovial membrane. Later, the membrane atrophies and the growth becomes free in the joint.

(4.) There may be cartilaginous outgrowths from the edges of the articular cartilages.

Rarely portions of the articular cartilages may be detached by violence or disease, or fibrinous concretions may result from synovitis.

PART III.

THE LESIONS FOUND

IN

CASES OF GENERAL DISEASES;

OF POISONING;

AND OF VIOLENT DEATH.

PYÆMIA.

UNDER the name of pyæmia are commonly understood several different conditions, giving rise to different lesions.

(1.) *Septicæmia*.—Some portion of the body is in a condition of gangrene. That is, the tissues are not only dead, but decomposing, with the evolution of gases, the softening and liquefaction of the solid parts, and the development of minute organisms, either animal or vegetable.

The gangrenous fluids thus produced are, apparently, absorbed by the lymphatics and blood-vessels, and are thus able to produce marked symptoms during life, and to cause death.

As in typhus fever, and other general diseases, hardly any lesions of the viscera are found, and the changes in the fluids have as yet escaped observation.

The bodies of persons, who have died from septicæmia, decompose with unusual rapidity. The endocardium and inner coat of the vessels are often stained with the coloring matter of the blood. The viscera are soft and flabby. The spleen may be swollen. The kidneys may be congested, their epithelium degenerated.

(2.) *Simple Pyæmia*.—Persons who have suppurating wounds or abscesses may, without much change in the wound or abscess, be seized with rigors followed by fever, become jaundiced and die.

In such persons, after death, no more lesions may be found than in septicæmia. The same tendency to decomposition, flabbiness of the viscera, and congestion of the spleen and kidneys is found.

Less frequently there may be purulent inflammation of the serous and synovial membranes, and of the connective tissue. Suppurative pleurisy, pericarditis, peritonitis, synovitis, and abscesses in the connective tissue may be found. Nor is there any way of accounting for these except by supposing that the pus from the original wound or abscess in some way infects the

system, and renders the tissues prone to take on purulent inflammation.

(3.) *Metastatic Pyæmia*.—This is a very different condition from the two preceding, and is accompanied with marked lesions. Either in consequence of wounds, injuries, inflammations, abnormal conditions of the system, or changes in the venous walls, the blood may become coagulated during life and form thrombi in the veins. These thrombi may become organized, or they may soften, break down, and their fragments be carried into the circulation. Some of such fragments ultimately become fixed in the small arteries and capillaries, and there exert a two-fold action. By their mechanical action in obstructing the vessels, they produce a collateral congestion, stasis, and extravasation of blood. By the irritative character of the thrombi themselves, they excite inflammatory action in the adjoining tissues. In this way are produced hæmorrhagic infarctions and multiple abscesses. The character of these infarctions will be more minutely described in connection with the organs in which they occur.

In persons, therefore, who die with metastatic pyæmia we find infarctions and small abscesses in the brain, lungs, heart-walls, liver, spleen, kidneys, and intestines. These infarctions very frequently produce inflammation of the serous coverings of the viscera in which they occur. The lungs are the organs most frequently involved. Further, we may find purulent inflammation of the serous and synovial membranes, and abscesses in the connective tissue. The emboli which produce the infarctions can sometimes be distinguished, sometimes not.

The thrombi, from which the emboli are detached, are sometimes readily found in the vein close to the original wound. In other cases, they may be in the vein at some distance from the wound; in other cases, it may be, in a neighboring vein, or even in one in some distant portion of the body. Sometimes no thrombus can be discovered.

Enlargement and congestion of the spleen and kidneys, and jaundice, are sometimes found.

THROMBOSIS AND EMBOLISM.

A thrombus is a clot, composed principally of the fibrine of the blood, formed during life in the cavities of the heart, the arteries, the veins, or the capillaries.

An embolus is a solid body, usually a portion of a thrombus, carried by the blood-current into some artery or capillary, and becoming fixed there.

A thrombus may entirely fill the cavity of a vessel; or it may only form a layer on its wall; or it may project from a smaller vessel into a larger one.

An embolus may completely, or only partly, plug up a vessel. It may remain alone, or a thrombus may be formed around it.

A thrombus may merely become harder and whiter with time; or it may become organized; or it may degenerate, soften, and change into a mass of puriform fluid and gangrenous-looking fibrine; or it may calcify.

The production of thrombi is due to various causes.

(1.) Spontaneous thrombi are produced by any cause which seriously weakens the whole system, as old age and chronic exhausting diseases. They are usually formed in the veins of the lower limbs and pelvis, and in the sinuses of the dura mater.

(2.) Compression of the vessels by ligatures, tumors, dislocated bones, and inflammatory exudation. Pneumonia may produce thrombosis of the pulmonary veins. Phthisis may produce thrombosis of the pulmonary arteries.

(3.) Dilatation of the vessels, as in aneurisms and varicose veins.

(4.) Thrombi may form in capillaries or small veins, and increase until they project into larger veins.

(5.) Inflammation, especially of a gangrenous and suppurative nature, in the soft parts around a vessel.

(6.) Degenerative and inflammatory changes in the wall of a vessel. In the arteries and capillaries, fatty degeneration and endarteriitis may render the inner coat rough. The veins may become inflamed in consequence of inflammation of the surrounding soft parts, or of wounds of their walls. The inflam-

mation involves the outer and middle walls of the vein, producing little abscesses and consequent necrosis of portions of the inner coat. Waldeyer says that there may be, without suppuration, an inflammation of the entire wall of a vein, resulting in the formation of minute elevations on its internal surface.

(7.) Emboli may produce thrombi about them and in the veins corresponding to the arteries which they obstruct.

The causes of embolism are also various.

(1.) Fragments of thrombi in the veins are the most frequent material of emboli.

(2.) Thrombi of the heart, vegetations on the valves and on the walls of the aorta.

(3.) Portions of the endocardium and of the inner coat of the aorta may degenerate and be detached from endarteriitis.

(4.) Portions of tumors which project into the veins, the pigmented bodies in melanæmia, and fat may form emboli.

In a considerable number of cases, it is impossible to find any source for the embolus. It must be remembered, however, in emboli, in connection with wounds, that not only the veins of the wounded region are to be examined, but also those of the pelvis. For, while there are no venous thrombi from the injury, there may be some from the decubitus and condition of the patient.

The consequences of thrombosis vary with the situation of the clot.

Thrombi in the arteries produce anæmia of the region supplied by the artery, or congestion from the collateral circulation. Later, there may be inflammatory changes, softening, or gangrene.

Thrombi in the veins produce inflammation in and around the vein, œdema, thickening of the skin, and connective tissue, gangrene, hæmorrhages.

Thrombi in the capillaries produce circumscribed congestions and hæmorrhages, (infarctions,) followed by degenerative and inflammatory changes, or there may be a more diffused gangrene.

The consequences of embolism vary with the character of the embolus and its situation.

A simple plug of fibrine in a vessel only produces changes in the circulation; but a plug from a gangrenous thrombus has a tendency to excite suppurative inflammation wherever it lodges. Again, an embolus may entirely, or only partially, occlude a vessel.

The size of the artery occluded, and the character of its collateral circulation, also lead to a variety of results. If a large artery is stopped, the part becomes anæmic; if a small one, there may be venous stasis from the collateral circulation.

Thus, if the large arteries supplying the extremities are occluded, we find anæmia, coldness, neuralgic pains, hyperæsthesia or anæsthesia, pricking sensations, cramps and paralysis of the muscles, and finally dry gangrene.

Emboli of the pulmonary arteries produce dyspnœa or sudden death; of the coronary arteries, sudden death; of the cerebral arteries, paralysis and white softening of the brain; of the retina, sudden blindness and subsequent atrophy of the optic nerve; of the mesenteric arteries, congestion and gangrene of the intestines.

If there is sufficient collateral circulation to partially supply the part with blood, we find evidences of chronic want of nutrition. The region affected becomes atrophied; its temperature falls; slight injuries excite chronic inflammation; the muscles undergo fatty degeneration; the bones necrose; the viscera degenerate; the fingers and toes undergo dry gangrene.

Emboli in the smaller arteries and capillaries produce the so-called hæmorrhagic infarctions.

We may therefore distinguish four classes of changes produced by emboli.

(1.) The embolus remains at the point where it has become fixed, and becomes organized without producing any lesions. This is the case when there are abundant arterial anastomoses.

(2.) The region supplied by the plugged artery undergoes necrobiotic changes, which take the form of softening, or of wet or dry gangrene. Such changes are seen in the brain, skin, and extremities.

(3.) The region supplied by the plugged artery becomes the seat of venous congestion and extravasation of blood. This

takes place when the obstructed artery is a small one, and is terminal, that is, has no arterial anastomoses beyond the situation of the embolus. These hæmorrhagic infarctions are seen in the lungs, spleen, kidneys, brain, eye, and alimentary canal.

(4.) Abscesses are produced. This takes place when the embolus is of a gangrenous and irritative nature. Large abscesses are only found in the lungs and liver. Minute abscesses are found in other organs, and are probably produced by capillary emboli.

The manner in which infarctions are produced has recently been studied afresh by Cohnheim. He has introduced little globules of wax into the circulation of frogs, and observed, under the microscope, the changes which take place when these little emboli become lodged in the branches of the lingual artery.

If the plug is lodged in a terminal artery, the artery beyond the plug, the corresponding set of capillaries, and the vein, are filled with stationary blood, or are partly empty. After a time, the vein belonging to this system of capillaries receives a backward current of blood, beginning from the larger vein, into which it empties, this larger vein being supplied by unobstructed arteries. The backward flow of blood continues until the vein, capillaries, and artery are filled and distended. Up to this point the mechanism of the changes is clear. The next step in the process is more difficult to explain. There is an escape of blood from the congested vessels, so that the tissue is infiltrated with blood. This can hardly be accounted for by the mere backward current of blood, for the pressure can be no greater in the congested vessels than in any other part of the venous system. Cohnheim explains the extravasation of blood by a change in the walls of the congested vessels. He found that, if ligatures were applied for a short time to terminal arteries, as in the frog's tongue, the rabbit's ear, the kidney and intestines, so that the system of vessels was for a time emptied of blood, and the ligature then removed, so that the blood could return, the result was an extravasation of blood, resembling an infarction. No changes could be detected in the walls of the vessels, and he assumes that the temporary withdrawal of blood from

the vessels in some way impairs the nutrition of their walls, so that when they are again subjected to the ordinary blood-pressure the walls rupture.

An embolus produces the same condition of affairs. A set of vessels are at first emptied of blood, and the nutrition of their walls thus impaired. After a time, these vessels are filled up from the veins, their abnormal walls can no longer resist the blood-pressure, and hæmorrhage takes place.

Cohnheim also asserts that when an abscess is formed by an embolus, the embolus is not lodged in a terminal artery, and produces no infarction. The embolus merely excites a suppurative inflammation by its pressure as an irritating body.

After an hæmorrhagic infarction is formed, the subsequent changes in it are of a degenerative character. The blood loses its coloring matter, and breaks down into a mass of granules, forming a dry, yellow, wedge-shaped mass. It may remain in this condition, gradually diminishing in size. Or, it may soften, and break down into a puriform fluid. Or, it may be surrounded by a zone of inflammation, or of gangrene.

Emboli from the left heart usually become fixed in the arteries and capillaries of the aortic system; emboli of the right heart, or portions of thrombi from veins, in the capillaries of the lungs; emboli from the portal system, in the capillaries of the liver. Not infrequently, however, we find infarctions of about the same date, and referable to a venous thrombus in both the pulmonary and aortic systems. This may take place in two ways. Emboli may be so small as to pass through the pulmonary circulation, and become fixed elsewhere; or, they lodge in the lungs, produce infarctions there, secondary thrombi are formed in the pulmonary veins, and from these, emboli are carried into the left heart and aortic circulation.

TYPHOID FEVER.

The lesions of typhoid fever are well marked and constant. They may be conveniently divided into two classes.

(1.) Those which are characteristic of the disease. To this

class belong the changes in the lymphatic follicles of the intestines, in the mesenteric glands, and in the spleen.

(2.) Those which are very frequently found with this fever, and yet are not peculiar to it. To this class belong the changes in the parotid and pancreas, the degenerations in the liver, kidneys, and voluntary muscles, thrombosis of the blood-vessels, infarctions, diseases of the lungs, and suppuration of the connective tissue in various places.

(1.) *The Intestines.*—The lesions of the intestines consist in an inflammatory enlargement, and subsequent degeneration of the lymph-follicles, whether as solitary glands or as Peyer's patches.

The process appears to begin with a catarrhal inflammation of the mucous membrane, accompanied or immediately followed by changes in the lymphatic follicles. The lesions in the lymphatic follicles begin early; they have been observed in persons who have died forty-seven hours after the commencement of the disease.

The increase in size of the agminated and solitary follicles may be rapid or gradual. The follicles may be only slightly enlarged, or may project so as to fill up the cavity of the intestine. The enlargement is usually more marked in the agminated than in the solitary follicles. Usually the whole of an agminated follicle will be enlarged, but sometimes only a part of it. If the enlargement is gradual, the different follicles which make up a Peyer's patch are enlarged, while the septa between them remain but little changed, and give the patch an uneven appearance.

The patches which are only moderately enlarged are of reddish or reddish gray color, are soft and spongy, and their edges blend gradually with the adjoining mucous membrane. The patches which are more intensely affected are of gray or brownish color, of firm consistence, and rise abruptly from the surrounding mucous membrane, or even overhang it like a mushroom. The largest patches are sometimes more than $\frac{3}{8}$ inch thick.

The enlargement and infiltration may spread from the patches to the surrounding mucous membrane, so that the

patches appear very large, a number of them may become fused together, and there may be even an annular infiltration entirely around the lower end of the ileum.

The infiltration of the agminated follicles may also extend outward into the muscular coat, and even appear beneath and in the peritoneal coat as small, gray, rounded nodules. This condition is usually found only with a few patches in the lower end of the ileum; sometimes in the cœcum and appendix vermiciformis. These little gray nodules usually correspond to diseased patches beneath them; but sometimes they appear to excite an inflammation of the peritoneum, accompanied by the production of numbers of similar nodules all over that membrane. Hoffmann describes a case in which the inflammation extended to the pleura with the production of similar nodules there.

The solitary follicles are affected in the same way as Peyer's patches. They may be hardly enlarged at all, or be quite prominent, or may be affected over a larger portion of the intestine than are the patches. Very rarely the solitary follicles are enlarged, while the patches are not at all or but slightly affected.

The inflammation and enlargement of the agminated and solitary follicles is followed by a healing process. The character of this process varies according to the intensity of the previous inflammation.

(1.) If the disease was mild, and the enlargement of the follicles moderate, the enlargement gradually disappears, and the follicles resume their normal appearance.

(2.) In moderate enlargements, the retrograde processes affect first the follicles, and leave the septa between them still swollen and prominent. This gives to the surface of a patch a reticulated appearance. After a time, however, the entire patch becomes flattened and uniform.

(3.) The solitary follicles, or the separate follicles of a patch soften, break down, and their contents are discharged with some attendant hæmorrhage. This leaves a bluish-gray pigmentation in the situation of each follicle. This pigmentation may remain for years.

(4.) In more severe types of the disease, the enlargement of the follicles ends in ulceration. This takes place in two ways.

(a.) The enlarged follicles soften, break down, and discharge their contents into the intestine. In this way are formed small ulcers. These ulcers increase in size by the same softening process, which gradually attacks their edges, and in this way ulcers of large size may be formed. The ulcers may extend outward only to the peritoneal coat, or ~~it~~ may extend to the peritoneal coat and terminate in perforation.

(b.) In the severest forms of the disease, considerable portions of the enlarged patches slough *en masse*, leaving a large ulcer with thick, overhanging edges. The slough may involve only the follicles, or it may extend to the muscular and even to the peritoneal coat. These ulcers also may afterward increase in area, and several of them may become joined together.

The ulceration, if the patient recover, cicatrizes, its edges become flattened, its floor is covered with a thin membrane covered with cylindrical epithelium.

Both forms of ulceration sometimes end in perforation. This takes place by the extension of the ulcerative process through the peritoneal coat, or by the rupture of the floor of the ulcer from some mechanical cause. Peritonitis and death are the usual events. In rare cases, however, the patient recovers, the perforation being closed by adhesions.

The minute changes which take place in the course of the intestinal lesions are as follows:

At first the blood-vessels around the follicles are dilated and congested, while the follicles are swollen, and the epithelium falls off. Then the follicles increase largely in size, and the increase is due to a new cell-growth. The new cells are in part similar to the lymphoid cells, which normally compose the follicles; in part are large rounded cells, 6-8 times as large as these, and containing several nuclei. The production of new cells is not confined to the follicles, but extends to the adjacent mucous membrane. In many cases, also, little foci of the same cells are found in the muscular, subserous, and serous coats. This increased number of cells compresses the vessels, and the parts become anæmic. Soon the cells degenerate, either by

granular degeneration of the individual cells, or by gangrene of a large part of a follicle. In either case, the degenerated portion is eliminated into the intestine, and leaves an ulcer, with its edges and floor infiltrated with cells. After this, the cell growth goes on, and the ulcer enlarges, or the cells are replaced gradually by connective tissue, and cicatrization follows.

The lesions we have described are found most frequently and most developed in the lower part of the ileum. They are not always, however, confined to this situation. Enlarged and ulcerated follicles may be found over the entire length of the ileum, and even in the jejunum. They may also extend downward, and be found in the colon, even as far down as the rectum. The same changes may also take place in the appendix vermiformis.

Besides the regular typhoid lesions of the intestines which have been described, we occasionally meet with others of a more accidental character.

Gangrene of the intestinal wall sometimes occurs. It most frequently involves a portion of the wall corresponding to an ulcer, but may also affect other portions where no ulcer exists. The process may terminate in perforation or in healing.

Diphtheritic Inflammation may attack the mucous membrane of either the large or small intestine. The mucous membrane between the typhoid ulcers is covered and infiltrated with croupous exudation.

Peritonitis of a mild type is a frequent accompaniment of the intestinal lesions. It appears to have but little influence on the course of the disease.

Severe peritonitis is usually due to perforation, less frequently to ulcers which reach the serous coat, but do not perforate. When there is infiltration of the serous coat with the typhoid new growth, the peritonitis may be accompanied by a production of little gray nodules of the same character throughout the peritoneum.

Infarctions of the spleen, inflammation of the ovaries, and perforation of the gall-bladder are sometimes the cause of peritonitis.

Hæmorrhage from the intestines is merely due to the inflam-

matory swelling and congestion, and is slight; or it is due to the ulceration of the follicles and consequent destruction of blood-vessels. In the latter case, the bleeding may be so profuse as to cause death.

Mesenteric Glands.—The mesenteric glands undergo the same changes as the follicles of the intestines, and are usually affected in a degree corresponding to the intensity of the intestinal lesions.

The glands are at first congested and succulent, then there is a production of lymphoid cells and large cells, as in the intestinal follicles, and the gland becomes enlarged, sometimes to an enormous extent. When the enlargement has attained its full size, the congestion diminishes, and the cells begin to degenerate. This degeneration may take place slowly, so that the gland returns to its normal appearance; or more rapidly, so that little foci of softened puriform material are formed. If these foci are small, they are absorbed, leaving a fibrous cicatrix; if they are large, they become dry, cheesy, and inclosed in a fibrous capsule.

The Spleen.—In nearly every case of typhoid fever, the spleen is enlarged. Sometimes, however, this lesion fails, and that without any satisfactory reason. In some cases, a preëxisting thickening of the capsule, or old adhesions, seem to explain the non-enlargement; but in others, no such obstacle exists.

The enlargement of the spleen commences soon after the beginning of the disease, increases rapidly until the third week, remains stationary for a few days, and then diminishes.

The organ is congested, of dark red color, and of firm consistence, while it is increasing in size. After it has reached its maximum size, its consistence becomes soft, and there is a considerable deposit of brown pigment. The enlargement appears to be due to the congestion, and to an increase of the normal elements of the spleen.

Infarctions of the spleen are occasionally found in typhoid fever, although they are by no means peculiar to it. They may cicatrize, or soften and cause peritonitis.

Rupture of the spleen is said to occur, in rare cases, without the presence of infarctions.

The second class of lesions comprises those which are frequently found with typhoid fever, but are not peculiar to it.

The Mouth.—A number of changes are found about this region. The follicles at the root of the tongue and the tonsils may be enlarged; the muscles of the tongue may undergo waxy and granular degeneration; gangrenous ulcers may attack the floor and sides of the mouth, and destroy large areas of tissue.

The Pharynx may be the seat of catarrhal or of diphtheritic ulceration, producing superficial and deep ulcers.

The Parotid is, in a moderate number of cases, the seat of an inflammation which tends to suppuration. In this process, both the glandular acini and the connective tissue between them are involved. Which of the two has the larger share in the process is still in dispute.

A slight enlargement and induration of the parotid and submaxillary glands is said by Hoffmann to be a frequent lesion, and to depend on increase of the gland-cells and dilatation of the follicles with their secretion.

The Pancreas undergoes changes similar to those in the salivary glands. It becomes at first swollen and red, then hard and grayish, then yellow. The vessels are at first congested; afterward, there is increase of the gland-cells; and lastly, degeneration.

The Liver may preserve its normal character, or may present changes.

In many cases, the organ will be found soft and flabby. Minute examination then shows that the liver-cells have undergone degeneration. They are filled with fine granules and small fat globules, and the degeneration may go on so far that the outlines of the hepatic cells are lost, and nothing but a mass of granules can be seen.

Less frequently, we find in the liver very small, soft, grayish nodules resembling those found in the peritoneum. They are situated along the course of the small veins, and there is, at the same time, a diffuse infiltration of lymphoid cells along the small veins. The nodules consist of lymphoid cells; they are often too small to be distinguished with the naked eye.

The Heart.—In a considerable number of cases, the muscular tissue of the heart is altered. The heart feels soft and flabby, is of grayish or brownish color, the muscular fibres are infiltrated with fine granules, and sometimes with brown pigment. Or, the heart is firm, but friable and easily torn; its cut surface glistens, and its muscular fibres present the waxy changes which will be described with the muscles.

Thrombi in the cavities of the heart, and vegetations adhering to the valves, are sometimes found. They may give rise to emboli in different organs.

The Larynx is very frequently the seat of catarrhal inflammation, with or without superficial ulceration. Less frequently diphtheritic inflammation occurs, and, in severe cases, may produce serious lesions: destructive ulcerations, perichondritis, and profuse hæmorrhage.

The Lungs.—The recumbent position of the patients sometimes leads to a long-continued congestion of the lungs, which renders them unfit to perform their functions. The lower lobes of the lungs, and sometimes the upper, will be found dark-colored, firm, infiltrated with blood, containing but little air. Microscopic examination shows the capillaries filled with blood, and the air-cells containing a very variable number of lymphoid cells.

Catarrhal bronchitis exists in a large number of cases. It may run its course as such, or it may become complicated with lobular pneumonia. This lobular pneumonia may go on to cheesy hepatization and be the commencement of phthisis.

Sometimes lobar, croupous pneumonia is found.

Infarctions are found in the lungs in all their various stages, apoplectic, bleached yellow and white, softened, and puriform.

Gangrene of the lungs occasionally is found, either associated with lobular pneumonia, with infarctions, or as an independent condition.

The Kidneys, in a large number of cases, present changes similar to those in the liver; that is, a parenchymatous degeneration. The kidneys are of medium size, their surface smooth, but the regular markings of the cortex are lost and largely replaced by

a white color. The epithelium, especially of the convoluted tubes, is granular and broken down.

Infarctions are not uncommon.

Small, gray nodules, like those in the liver and peritoneum, are seen in a few cases.

The Ovaries.—Hæmorrhage and gangrenous inflammation have been observed in rare cases.

The Brain.—Edema of the pia mater and of the brain tissue is found in a certain number of cases. As to its frequency, authorities differ.

Acute meningitis, white softening, softening with capillary apoplexies and infarctions, are found in occasional cases.

The Voluntary Muscles undergo various changes.

(1.) The muscular fibres become filled with fatty and albuminous granules to such an extent that the markings of the fibres disappear. This degeneration is observed in other diseases.

(2.) The muscular fibres undergo a peculiar change, called by Zenker "waxy degeneration." It is not to be confounded with the "waxy" or "amyloid" degeneration of the liver, kidneys, etc. This change is also found in typhus, cholera, acute tuberculosis, small-pox, and other diseases, and in connection with injuries of the muscles. It is most common, however, in typhoid fever, and at the acme of the disease.

The change is usually found in the abdominal muscles, the adductors of the thigh, the pectoral muscles and diaphragm, and in the tongue.

The muscles appear to the naked eye of a grayish or yellow color, dry, brittle, with a wax-like section, resembling fish muscle. Microscopic examination shows the muscular fibres to be converted into a peculiar, homogeneous, highly refracting substance. According to the manner in which a fibre is attacked, we find long, wax-like cylinders, or smaller, irregular bodies of very various shape. The change in the fibres is accompanied or followed by a new growth of muscle. We find then numbers of lymphoid and fusiform cells with large fusiform bodies, and large, flat, plate-like bodies, each containing many nuclei, and some of them distinctly striated.

The entire process is differently interpreted by different observers. Some hold that it is a degeneration followed by atrophy and a new growth. Others, that the new growth precedes and causes the degeneration. Others, that, instead of a degeneration, the waxy appearance is produced by a coagulation analogous to that which produces the rigor mortis, and that the entire appearance is a post-mortem one.

(3.) Rupture of the muscles is apparently produced by their waxy degeneration. It is accompanied by hæmorrhage, sometimes to a large amount. It may cicatrize, or become the seat of a purulent, gangrenous inflammation.

(4.) Suppuration of the muscles or of their fasciæ is occasionally seen ; it may produce large abscesses.

The Skin.—Gangrenous inflammation of the skin frequently occurs in the form of bed-sores, affecting the skin over the sacrum and trochanters, where it is subjected to the constant pressure of the bed. Gangrene of other portions of the integument, especially of the toes, is produced by thrombosis or embolism.

There may be suppurative inflammation of the connective tissue in various parts of the body. Perhaps the most important is that producing retro-pharyngeal abscesses.

BILIOUS TYPHOID.

Under this name Griesinger describes a form of fever resembling in its symptoms both relapsing and typhoid fever, and attended by very grave symptoms.

The Skin is jaundiced ; sometimes ecchymotic.

The Brain is usually anæmic. The pia mater may contain hæmorrhages.

The Pharynx and Larynx may be the seat of catarrhal, croupous, and ulcerative inflammation.

The Lungs.—Catarrhal bronchitis, lobar, and lobular hepatization, and infarctions are not infrequent.

The Heart is usually flabby and pale.

The Liver is enlarged and congested, or anæmic, flabby, yellow, and fatty. Its surface is often coated with fibrinous exudation.

The Spleen is often coated with exudation. It is always enlarged, congested, soft, and may undergo spontaneous rupture. In most cases, it is filled with countless small, grayish nodules—the enlarged Malpighian bodies. These afterward soften and form little abscesses. Infarctions are not uncommon.

The Stomach and Intestines are often the seat of catarrhal and croupous inflammation. The solitary and agminated follicles are not enlarged.

The Mesenteric Glands and the other lymphatic glands in the abdomen are usually enlarged.

The Kidneys are frequently congested, or anæmic, and the seat of parenchymatous degeneration.

TYPHUS FEVER.

The lesions of typhus fever are variable and not very characteristic.

The Skin.—The regular eruption of typhus disappears after death, but sometimes small ecchymoses remain. Gangrene from bed-sores, and of the toes from thrombosis, are sometimes seen.

Decomposition commences early, and the blood is unusually fluid and prone to stain the vessels.

The Brain usually presents no lesions. Acute meningitis is said sometimes to occur.

The Pharynx and Larynx may be the seat of catarrhal or diphtheritic inflammation. Gangrenous inflammation of the walls of the mouth may occur. Suppurative inflammation of the parotid glands is not uncommon.

The Lungs.—Bronchitis frequently exists. Hypostatic congestion, lobular and lobar pneumonia and infarctions are sometimes found.

The Heart is soft and flabby. Its muscular fibres are often filled with granules.

The Spleen is usually large and soft. There may be infarctions.

The Liver and Kidneys may present the same parenchymatous degeneration as is observed in typhoid fever.

The Intestines may appear normal, or the agminated and solitary follicles may be slightly enlarged, or there may be catarrhal or diphtheritic inflammation.

The Voluntary Muscles sometimes undergo the same granular and waxy degeneration as in typhoid fever.

RELAPSING FEVER.

The lesions of relapsing fever are neither characteristic nor constant.

In some cases, no marked lesions are to be found.

The Liver and Spleen are frequently enlarged.

The Lungs may be congested or hepatized in the lobular or lobar forms.

The Kidneys may be congested, or may undergo parenchymatous degeneration.

Hæmorrhages in the walls of the alimentary canal, the air-passages, and in the skin are sometimes found.

MALARIAL FEVERS.

INTERMITTENT AND REMITTENT FEVER.

The lesions of malarial poisoning are usually characteristic and well marked. Their extent varies with the intensity and duration of the disease and the period at which the patient dies.

Intermittent Fever corresponds to a mild degree of malarial poisoning, and seldom causes death.

Remittent Fever, especially the "pernicious" form, corresponds to an intense degree of the poison, and frequently causes death.

The Blood.—The most constant lesion is the presence of little particles of black or reddish pigment in the blood. The pigment is in the form of flakes, or of granules imbedded in small, irregular, transparent, finely-granular bodies. We also find these same transparent bodies without pigment, some of them looking like round and fusiform cells. Sometimes there is an increase of white blood-globules.

The presence of this pigment in the blood gives to the different organs of the body a peculiar, dark color, varying in shade according to the proportion of pigment. Sometimes the change in color is not perceptible to the naked eye, and only microscopic examination shows the pigment in the vessels. The pigment is distributed in very different amounts in the different organs.

The Spleen is the organ in which the pigment is found in the greatest amount and most constantly. Here it is difficult to ascertain whether the pigment is entirely in the vessels, or also in the parenchyma. The spleen will be found of reddish brown, olive, or black color. It is usually enlarged, sometimes enormously so; its consistence may be normal, or soft, or firm. In mild intermittent fever, the spleen may be much enlarged and not pigmented.

The Liver is almost as frequently and intensely pigmented as the spleen. It is of a light or dark brown, or olive, or black color. The pigment will be found in the venous and arterial capillaries and small veins. Sometimes, while the change in color of the liver is so slight as hardly to be appreciated, a small amount of pigment will still be found in the capillaries. The liver may be of normal size, or large and congested, or small. The hepatic cells usually appear perfectly normal. In some cases there is intense congestion, and even hæmorrhages, in the substance of the organ.

The Brain is much less frequently affected. In marked cases, the gray matter of the convolutions and ganglia appears of a dark gray color, contrasting strongly with the white matter. Sometimes there are multitudes of little capillary apoplexies.

The pigment will be found in the small vessels and capillaries of the pia mater and gray matter. It may accumulate in the vessels so as to obstruct them, and produce the capillary apoplexies just mentioned.

In other parts of the body the pigmentation is usually not sufficient to be seen with the naked eye. Careful microscopical examination, however, will very often detect pigment in the

small vessels and capillaries of the kidneys, intestines, lungs, skin, and connective tissue throughout the body.

If the patient die during a chill, all the organs are usually intensely congested.

The Stomach and Small Intestine may be the seat of catarrhal inflammation; the large intestine, of croupous inflammation; jaundice is not uncommon.

Œdema of the feet and legs, ascites, and general anasarca, or ascites alone, may be found after only a few paroxysms of the disease. The cause of this condition is not apparent.

Persons who have become anæmic and cachectic from long continuance of the disease may develop œdema in the same way as other anæmic persons.

YELLOW FEVER.

The Skin and other tissues are usually jaundiced; the color ranges from light yellow to dark mahogany. Petechiæ and ecchymoses are often found. The muscles sometimes contain large hæmorrhages.

The Brain and Spinal Cord present no marked lesions.

The Lungs.—Catarrhal bronchitis, hæmorrhagic infarctions of the lungs, and ecchymoses of the pleura are sometimes found.

The Heart.—The pericardium may contain serous and fibrinous exudation or blood. There may be ecchymoses in the pericardium. The heart is frequently soft and flabby, its muscular fibres far advanced in fatty degeneration.

Alimentary Canal.—There may be deep erosions of the mucous membrane of the œsophagus. The stomach and small intestines usually contain blackened blood. The mucous membrane of the stomach may be normal, or congested, with ecchymoses and hæmorrhagic erosions. The mucous membrane of the stomach may also be the seat of catarrhal inflammation, with swelling of the solitary and agminated follicles.

The Liver may be normal or hyperæmic. Most frequently, however, by the third day of the disease it presents peculiar changes. It is of normal size, or slightly enlarged, or atrophied. It contains little blood, is of a *café au lait* or yellow

color; its consistence is flabby and dry. The hepatic cells are filled with granules, or with fat globules.

In persons who have recovered from the fever and died a few weeks later from other causes, the liver has been found entirely normal.

The Spleen is normal, or enlarged and congested.

The Kidneys are frequently the seat of a parenchymatous degeneration analogous to that of the liver.

CHOLERA.

In this disease the lesions do not by any means correspond to the severity of the symptoms during life. This is especially the case in mild epidemics of the disease, and in cases which die during the stage of reaction. In many such cases there are no marked lesions.

If death occurs during the invasion of the disease, or in collapse, in severe cases the appearances are as follows:

The bodies remain warm for some time, and the temperature may even rise for a short time after death. The rigor mortis commences soon, and lasts unusually long. The muscles sometimes exhibit a peculiar spasmodic twitching before the rigor mortis sets in, especially the muscles of the hand and arm.

The Skin is of a dusky gray color, the lips, eyelids, fingers, and toes of a livid purple. The cheeks and eyelids are fallen in, and the face has a peculiar sunken look.

The Brain.—The sinuses of the dura mater are filled with dark, thick blood. The pia mater may be normal, or œdematous, or ecchymosed, or covered with a thin layer of fibrinous exudation. The brain is usually normal.

The Lungs.—The pleura is often covered with a thin layer of fibrinous exudation. The lungs are retracted and anæmic.

The Heart is unchanged.

The Peritoneum is often covered with a layer of fibrinous exudation.

The Stomach is usually unchanged, sometimes the seat of catarrhal inflammation.

The Small Intestine may appear normal, or anæmic, or con-

gested in various degrees. The mucous membrane may be soft and œdematous. In the intestines we find a variable quantity of the rice-water fluid seen in the evacuations during life. The solitary and agminated follicles may be much swollen.

The Large Intestine is usually normal. In some epidemics, however, it is prone to croupous inflammation with ulceration.

The Liver and Spleen undergo few changes; they may be anæmic and flabby.

The Kidneys appear normal, or large and congested, or with white and thickened cortex. The tubes contain degenerated epithelium and casts.

The Uterus and Ovaries may be congested, and contain hæmorrhages.

If the patient does not die until after the acute symptoms of the disease have passed by, the appearances are different.

The body does not present the same collapsed appearance.

The Larynx may be the seat of purulent or diphtheritic inflammation.

The Lungs.—There may be pleurisy with sero-purulent exudation. The lungs may be congested and œdematous, or contain infarctions, or lobular hepatizations, or there may be bronchitis.

The Stomach and Intestines may be the seat of croupous inflammation.

EPIDEMIC CEREBRO-SPINAL MENINGITIS.

This disease presents itself under a variety of forms in different epidemics, and in different localities. The severity of the disease, its duration, and its symptoms are all variable. Its lesions also are not constant. Those which will be enumerated below are not all or any of them necessarily present in every case:

The Skin may be unchanged, may present petechial spots, or large purpuric patches. In many cases vesicles of herpes are found on the face and elsewhere.

The Muscles frequently are the seat of granular degeneration. Sometimes there is an increase of cells between the muscular fibres, which may go on to the formation of abscesses.

The Rigor Mortis is pronounced and long continued, but decomposition sets in early.

The Skull.—The bones of the skull may be hyperæmic.

The Brain.—A few cases are reported in which no lesions of the brain or its membranes were seen ; but, as a rule, lesions are found. The dura mater may be covered on its inner surface with a layer of fibrinous exudation and extravasated blood ; or it may be adherent to the pia mater. There may be purulent matter between the dura and pia mater.

The pia mater is usually congested. Infiltrated in it and beneath it we find serum, gelatinous matter, and pus in variable amounts. There may be a general opacity and thickening of the pia mater, or there are scattered purulent foci. These changes are most frequent and most marked at the base of the brain. Microscopic examination of the pia mater, where it appears unchanged, often shows an increase of cells. The gelatinous matter beneath and in the pia mater appears to be made up of pus imbedded in a mucous basement substance.

The ependyma of the ventricles may contain small ecchymoses. The walls of the ventricles may be softened and cedematous. There may be pus in the ventricles and in the choroid plexuses. Sometimes the ventricles are enormously distended with serum.

In the brain tissue there may be capillary apoplexies, large apoplexies, general hyperæmia, spots of white softening, cedematous softening, suppurative encephalitis, infarctions, granular softening around the small vessels, red softening, cell-growth in the gray matter of the cortex.

Spinal Cord.—There may be small extravasations of blood in the loose tissue outside of the dura mater. The dura mater may be congested, its internal surface covered with fibrinous exudation, or it may be adherent to the arachnoid. There may be pus, serum, or gelatinous matter between the dura and pia mater. The pia mater is congested, infiltrated with pus, serum, and gelatinous matter. The same substances are found beneath it. The exudation is found on the posterior aspect of the cord rather than on its anterior. The inflammatory lesions may extend the entire length of the cord, or only over part of it, or

over discontinuous portions. The central canal of the cord may be dilated and contain pus. The cord may be softened. There may be a cell-growth, or a production of pus about the roots of the spinal nerves.

The Ears.—The auditory nerves may be œdematous, softened, or imbedded in pus. There may be suppurative inflammation of the internal ear.

The Eyes may be the seat of suppurative choroiditis.

The Lungs.—The mucous membrane of the bronchi may be congested. The lungs may be congested and œdematous; or there may be hepatization of larger or smaller portions of the lung tissue.

The Stomach.—There is said to be sometimes an unusual degree of softening of the fundus.

The Intestines.—The solitary and agminated follicles may be swollen and even ulcerated.

The Spleen may be larger, or small, or pigmented.

The Liver is usually of medium size. The hepatic cells are often the seat of granular degeneration.

The Kidneys.—The epithelium of the tubes may be the seat of granular degeneration.

SCURVY.

The bodies decompose early. The skin is of a dusky color, sometimes œdematous on the legs, mottled with a variable number of small and large, purple, blue, brown, or blackish spots, produced by extravasations of blood in the superficial layers of the cutis.

The gums are swollen, soft, and livid, the teeth loose, the alveolar walls often necrotic.

In the subcutaneous connective tissue, and in the muscles, we may find extravasations of blood, sometimes of very large size, in various stages of absorption, induration, and softening. Sometimes these extravasations produce perforation of the skin and ulcers.

The joints may be inflamed, may contain serum or blood. Rarely, the hæmorrhages are followed by destruction of the cartilages and ends of the bones.

Very rarely, there may be hæmorrhage between the periosteum and bone, and in the bone itself, producing softening and destruction of the bone, and separation of the epiphyses. The sternal ends of the ribs are the most frequent seat of this change.

The pleura, pericardium, and peritoneum may be the seat of ecchymoses, or contain large hæmorrhages in their cavities, or be coated with fibrinous exudation.

The lungs may be congested, œdematous, or the seat of lobular pneumonia.

The mucous membrane of the air-passages usually contains ecchymoses.

The spleen is swollen, soft, and may contain infarctions.

The mucous membrane of the stomach and intestines is swollen, congested, contains hæmorrhages, exhibits small hæmorrhagic ulcers, and may be coated with blood.

The kidneys may appear normal, or may be in various stages of Bright's Disease.

ADDISON'S DISEASE.

Under this name is usually understood a disease characterized by a dark-colored or bronzed skin, by disease of the supra-renal capsules, and by a peculiar group of symptoms. The symptoms are: A change in the mental condition; great anæmia, with increase of the white-blood globules, but without emaciation; frequent and protracted syncope; great prostration; pains in the back and epigastrium; dyspepsia and vomiting; diarrhœa; sometimes convulsions, neuralgia, and paraplegia; typhoid symptoms and death.

The pigmentation of the skin is the symptom which has principally excited attention. The change in color usually begins, and becomes most intense in those parts of the skin which are not covered by the clothing. The rest of the skin afterward changes color, but not uniformly; for white patches are often left. The color is at first a light yellow or brown; this becomes darker until it is a dark greenish brown, grayish or blackish brown. There may be black spots on the mucous membrane of the lips and mouth.

Unfortunately, under the same name of Addison's Disease, different observers have described cases in which the symptoms and bronzed skin existed without disease of the supra-renal capsules; cases in which the bronzed skin was the only lesion; cases in which the supra-renal capsules were diseased without symptoms or bronzed skin.

In the present state of our knowledge, we have to admit that the connection between the color of the skin, the condition of the supra-renal capsules, and the symptoms is an unsolved problem. Each of the three conditions, the symptoms, the colored skin, and the diseased capsules may exist separately, and yet in a very considerable number of cases they are combined.

I can only, therefore, give such lesions as have been observed, without implying that they are the universal or necessary lesions of the disease.

The Skin.—The pigmentation resembles that of the negro. It is a diffuse or granular pigmentation of the inner layers of the rete Malpighii. Sometimes pigment is found along the nerves and vessels in the cutis.

The Brain.—Softening, hydrops of the ventricles, pigmentation of the gray matter, exudation on the arachnoid, hyperæmia and anæmia of the brain, chronic inflammation, degeneration of the hypophysis cerebri, and concretions in the medulla oblongata have all been observed.

The Sympathetic Nerves.—Atrophy, fatty degeneration, hyperæmia, pigmentation, and fibrous degeneration, especially affecting the abdominal portion of the sympathetic, have been described.

The Supra-renal Capsules.—The most frequent lesion is produced by a chronic inflammation with cheesy degeneration. Less frequently, tubercles, carcinoma, apoplexy, fatty and waxy degeneration have been observed.

The most striking cases are those in which at the autopsy no other lesions exist, nor can any other cause for death be found than the change in the supra-renal capsules.

LEUKÆMIA.—PSEUDO-LEUKÆMIA.

Under these two names, we understand two diseases, in which are found the same lesions of the viscera, but in which the condition of the blood is different.

The lesions consist in the new growth of lymphatic glandular tissue in different parts of the body.

This new growth takes place as an hyperplasia of the spleen alone, or of the lymphatic glands alone, or of both together; or there may be also small growths of the same nature in the tonsils, larynx, and trachea, lungs and pleura, stomach and intestines, pericardium, liver, spleen, and kidneys. The lymphatic glands in every part of the body may be enormously enlarged, but do not become inflamed, softened, or cheesy. The growths in the different viscera will be found fully described under their respective headings.

With these lesions in the viscera, there may be also a very marked increase in the number of the white blood-globules in leukæmia; or the proportional number of white-globules may be very slightly, or not at all, increased in pseudo-leukæmia.

GOUT.

The characteristic lesion of gout is the deposit of urate of soda in the articular cartilages, the ligaments of the joints, the ears, and the eye-lids.

The most frequent situation is the metatarso-phalangeal joint of the great toe. The cartilage may be infiltrated or incrustated with the deposit.

If the gout is of long standing, there is usually chronic diffuse nephritis. The kidneys are small, the cortical substance atrophied, the surface irregular, the texture hard, the pyramids marked by white lines running parallel to the tubes. These white lines consist of urate of soda.

GLANDERS.—FARCY.

These two names are given to two varieties of the same disease. The disease originates in the horse, and occurs in

men by contagion. According to some authors, it may be idiopathic in the human subject.

In the horse, we find four varieties of the disease: Chronic and acute glanders, chronic and acute farcy.

(1.) *Chronic Glanders*.—The disease begins in the mucous membrane of the nose. Small, whitish nodules, composed of small round cells, are formed in the mucous membrane. These nodules soften and ulcerate. The same nodules may be found in the larynx, trachea, and bronchi. The ulcerations may remain superficial, or they may extend and attack the subjacent cartilage and bone. Nodules of the same kind may be found in the lungs.

(2.) *Acute Glanders*.—There is the same formation of nodules, which soften and ulcerate; but they are accompanied with an intense inflammation of the nasal mucous membrane, and the disease runs a rapid course.

(3.) *Chronic Farcy*.—The lymphatic glands and vesicles become enlarged, and nodules are formed in the skin, lungs, and other viscera. The glands become cheesy, or soften and suppurate. The cutaneous nodules soften and suppurate.

(4.) *Acute Farcy*.—There are the same lesions of the lymphatics, and nodules and abscesses are found in the skin. There are also nodules in, and inflammation of, the nasal mucous membrane, and the disease runs an acute course.

In man, the disease occurs in an acute and chronic form, but does not exactly resemble any of the varieties of the disease in the horse.

The acute disease runs a rapid and malignant course. The skin may be covered with a pustular eruption. Furuncles, carbuncles, and abscesses beneath the skin and in the muscles are found. Nodules are found in the nasal mucous membrane, the lungs, kidneys, testes, spleen, and liver. The joints may be inflamed, and there may be osteo-myelitis.

The disease may begin at a single point, so that it may be mistaken for a carbuncle, or a gangrenous erysipelas. Graefe gives a case which began as an acute exophthalmos, and the nature of the disease was not ascertained until after death. There were little nodules in the choroid coat of the eye.

The chronic form of the disease is still more difficult of diagnosis.

The nodules grow very slowly, are hard, and may occur in groups, or like a string of beads.

The nodules may soften and form deep, chronic ulcers.

HYDROPHOBIA.

There are no characteristic lesions in this disease.

Congestion of the nervous centres, and congestion and swelling of the mouth and pharynx, are observed in some cases. Decomposition usually commences soon after death.

SUNSTROKE.

During the hot summer months, cases of sunstroke are of frequent occurrence in New-York. The persons affected are, for the most part, adult male laborers, usually of intemperate habits.

It is necessary to separate from the cases of sunstroke proper, when the patient is attacked while exposed to the heat of the sun, the cases of exhaustion from heat and fatigue, which may occur as well in the house.

The patients who are seriously affected by sunstroke, exhibit, during life, an intense heat of the skin, convulsions, and coma. Death in many cases soon ensues.

After death, decomposition sets in very early, owing to the state of the weather. In autopsies which I have made within two hours after death, the increased heat of the skin was still maintained.

The Brain and its membranes were in some cases congested ; in others not. Sometimes there was an increased amount of serum beneath the pia mater ; sometimes there were small and thin extravasations of blood beneath the pia mater, and between the pia and dura mater.

In the other viscera there were no lesions except those due to the condition of coma, existing before death. The lungs and kidneys were frequently congested.

TRICHINIASIS.

The *Trichina spiralis* is found in man, the pig, the dog, the cat, the rabbit, the guinea-pig, the rat, and in some birds, both in its undeveloped and in its fully developed form.

In its fully developed form it is found in the small intestines, and less abundantly in the large intestine. It is a viviparous worm, with a long, rounded body, sharp at the head, rounded at the tail. The males are 1.5 mm. long, the females 2-3 mm. long. The intestinal canal runs the entire length of the body. The female organs of generation are simple, and placed for the most part in the second third of the body. The testicles are in the last third of the body.

In its undeveloped form, the worm resembles a hair, is about 1 mm. long, and possesses no organs but an alimentary canal. It is rolled in a spiral form. From one to three worms are enveloped in a capsule, which is at first thin and membranous, afterward calcified. In this condition they are scattered in immense numbers throughout the voluntary muscles, and may remain so for an indefinite number of years.

After a man has eaten uncooked flesh containing the undeveloped trichina, the worms remain in his intestines, and by the second day have become fully developed and freed from their capsules. The females are impregnated, and by the eighth day bear immense numbers of young worms. By the tenth day, the young worms find their way through the connective tissue, and by the blood into the voluntary muscles. By the fourteenth day we usually find them encapsuled throughout the muscles. The muscular fibrillæ become homogeneous, or break down into granular matter; the sarcolemma is thickened, and contracts at each end so as to form a cavity for the worm. Within this portion of the sarcolemma the cyst is formed by a hardening and calcification of the granular substance. At the same time there is a growth of lymphoid cells about the affected fibrillæ. New capillaries are formed about the capsule, and new muscular fibrillæ are formed to take the place of those which have been destroyed. (Wagner.)

I have seen in one case, in which death took place on the four-

teenth day after eating the infected flesh, the trichinæ free between the muscular fibrillæ, and at the same time an abundant production of lymphoid cells in the interstitial connective tissue.

We most frequently find the parasites with calcified cysts in persons of whose history we know nothing. But occasionally we meet with acute cases in which death has ensued in a short time from the effects produced by the trichina.

DEATH FROM SUFFOCATION.

By suffocation we understand that condition in which air is prevented from penetrating into the lungs. Many deaths from drowning and strangulation take place in this way.

The methods in which the supply of air may be cut off from the lungs are very various. The mouth and nose may be closed by the hand, by plasters and cloths, by wrapping up the head in cloths, by covering the face with earth, hay, grain, etc. Foreign bodies may be introduced into the mouth, pharynx, and larynx. Blood may pass into the trachea from an aneurism, or from a wound. The glottis may be closed by inflammatory swelling. Matters which are vomited may lodge in the larynx.

External Inspection.

The body should be examined for marks of violence, the cavities of the mouth and nose for foreign substances.

The face may be livid and swollen, or present a natural appearance. The conjunctiva may be congested and ecchymotic. There may be small ecchymoses on the face, neck, and chest. The mouth often contains frothy blood and mucus. The tongue may be protruded.

Internal Examination.

The Brain and its membranes may be congested, or anæmic and œdematous, or unchanged.

The Blood throughout the body is unusually dark-colored and fluid.

The Larynx may contain foreign bodies which have produced

the suffocation. The mucous membrane of the larynx, trachea, and bronchi is congested and sometimes ecchymotic. These passages contain frothy blood and mucus.

The Lungs are usually congested and œdematous, but sometimes do not differ from their ordinary appearance. There may be small patches of emphysema on the surface of the lungs. Sometimes, especially in infants, small ecchymoses are found in the costal and pulmonary pleura.

The Heart usually presents its right cavities full of blood, its left cavities empty.

The Abdominal Viscera are usually congested.

DEATH FROM STRANGULATION.

Strangulation is effected by the weight of the body in hanging; by pressure on the neck with the hands; or by constriction of the neck by a cord of some kind. Death is usually produced by the pressure on the larynx and trachea; that is, by asphyxia. In some cases, death appears to be caused by pressure on the large vessels of the neck; and, in some cases of hanging, by fracture or dislocation of the cervical vertebræ.

External Inspection.

The face may be livid and swollen, the eyes prominent, the lips swollen, and the tongue protruded. These appearances, however, are not the rule. The greater number of those who die from strangulation cannot be distinguished by their faces from persons who have died in any other way.

Erection of the penis, ejaculation of semen, and evacuation of fæces and urine, are appearances on which some stress has been laid, but they do not appear to be constant or characteristic.

In almost all cases, there are marks left on the neck by the implements which have produced the strangulation.

In cases of suicidal hanging, the cord usually encircles the neck between the chin and the os hyoides. The neck is but little compressed, while the cord leaves its mark behind the ears and on the sides of the occiput. When strangulation is

effected by a cord or cloth drawn around the neck, the latter will bear a mark nearly encircling it.

In both cases, the mark may be one or two lines deep, or very superficial. It corresponds in width to the cord which has produced it. It is of a dirty yellow color, hard, and with the epidermis partly rubbed off; or it is of a bluish-red color and soft; or it is soft and not discolored; or, very rarely, there are ecchymoses beneath it. The same mark may present all these appearances in different parts of its course.

In cases of strangulation by the fingers, the impressions left by them present the same appearances, but correspond in shape to the fingers.

Exactly the same appearances are seen in bodies which have been hung or strangled soon after death.

Internal Examination.

The Brain and its membranes may be congested, or there may be extravasation of blood, or there may be no abnormal appearances.

The Neck.—In some cases, we find rupture of the cervical muscles, fracture of the os hyoides, fracture of the cartilages of the larynx, rupture of the intervertebral ligaments, fracture and dislocation of the cervical vertebræ, rupture of the inner and middle coats of the carotid arteries. Similar lesions can be produced in dead bodies, but only by the use of great violence.

If death takes place from asphyxia, the larynx and trachea are congested and full of frothy blood and mucus; the lungs are congested and œdematous; the right cavities of the heart are full of blood; the abdominal viscera are congested.

There are cases of strangulation in which the results of the post-mortem examination are entirely negative.

DEATH FROM DROWNING.

In examining the bodies of persons who have been drowned, it is necessary to bear in mind a number of questions which may arise: Whether the person came into the water alive or dead; how long a time has elapsed since death; whether the

person committed suicide, or was drowned by accident, or was murdered. These questions are to be solved sometimes certainly, sometimes with probability, sometimes not at all by the post-mortem examination.

External Inspection.

It has been asserted that the bodies of drowned persons are colder than the average. The point has not, however, been determined by any exact thermometrical observations.

The peculiar condition of the skin called *cutis anserina* is found very constantly before decomposition has set in, except in the case of new-born infants. This same appearance, however, is frequent in persons who have died in any sudden and violent manner.

Analogous to this is the retraction of the penis and scrotum in the male, and of the nipples in the female. Casper says that such a retraction is almost always present.

We sometimes find sand, earth, weeds, etc., still grasped in the hands, or remaining under the nails.

The rigor mortis appears to come on quickly; so that the body and limbs may be found still in the convulsed and distorted attitude which they assumed at the time of death. When the rigidity has passed off, the limbs become relaxed.

After the body has been in water for 12–24 hours the hands and feet become of a livid, grayish-blue color. After two or three days this change of color becomes still more marked, and the skin is wrinkled in long folds.

In new-born infants the remains of the umbilical cord will not be found dried and mummified, unless the child was dead before it was placed in the water, or unless it was three or four days old before it was drowned.

Decomposition takes place sooner in summer than in winter, sooner in stagnant water than in a running stream. As soon as a body is removed from the water to the air, putrefaction goes on much more rapidly. In bodies which have lain in the water decomposition begins in the face and head and goes downward, instead of its usual course of first appearing in the abdominal wall.

The face, scalp, and neck are of a bluish, livid color, which soon changes to a red. The red next becomes mottled with greenish spots. The greenish spots become larger, and the discoloration extends down on to the thorax. The head and neck are swollen, the eyelids, nose, and lips project, extravasated blood, either fluid or coagulated, is found in the subcutaneous tissue. In this decomposed and softened condition, it is often very difficult to distinguish between the effects of ante-mortem violence and those of post-mortem putrefaction. Later the scalp is detached from the bones. The entire body is black and enormously swollen; the nails drop off; the joints are separated; the muscles are saponified; the cavities of the abdomen, thorax, and head are open.

Internal Examination.

Persons who have been drowned usually die from asphyxia. Sometimes, however, they die in some unknown way, without any evidence of asphyxia. These latter cases are said to die from syncope, or from neuro-paralysis. The lesions are correspondingly different.

The Brain.—Congestion of the brain and its membranes is only found in a small proportion of cases. Extravasation of blood has only been observed in a very few cases.

The Air-Passages.—In persons who die from asphyxia the mucous membrane of the larynx, trachea, and bronchi is usually congested, the mucous glands are distended and appear like little vesicles, a quantity of frothy blood and mucous fills the air-passages.

In those who die from syncope, these appearances are absent.

Foreign substances from the water, and matters regurgitated from the stomach, may find their way into the air-passages during the act of drowning, or as a post-mortem occurrence.

The Lungs usually are much increased in size, so that they entirely fill the thorax and cover the heart. The increased size is due partly to the distention of the air-vesicles with air, and partly to the presence of the fluid in which the person was drowned. The fluid is inspired in the act of drowning. The

lungs are usually congested, if the death is from asphyxia. They do not crepitate well, but have a spongy feeling.

The Heart.—In those who die from asphyxia, the right cavities of the heart are filled with blood, while the left cavities are empty.

The Blood remains unusually fluid throughout the body, and is of a cherry-red color.

The Stomach.—The fluid in which the person is drowned, mixed with earth, weeds, etc., may be swallowed during the act of drowning, or may find its way into the œsophagus and stomach after death.

The Abdominal Viscera may all be congested in those persons who die from asphyxia.

In those persons who do not die from asphyxia, but from syncope or neuro-paralysis, we find no lesions.

DEATH FROM BURNING.

Death may be caused by the inspiration of smoke and flame; by the immediate effect of the burning; by secondary affections of the viscera; and by the exhaustion produced by the long continued inflammation and suppuration of the skin.

The entire body may be burnt to a coal, or completely roasted, or only a larger or smaller area of the skin be burned.

We find the burned skin divested of epidermis, and presenting a peculiar red, hard, parchment-like appearance. If the patient has lived some time, this is replaced by a suppurating surface. Or there are small, bladder-like elevations of the epidermis. The base of these blisters is red, and they are surrounded by a red zone, or suppuration may have commenced.

These appearances cannot be produced by heat applied to the skin after death.

The Brain may be congested, œdematous, or softened. More frequently it is normal.

The Larynx and Trachea may be the seat of croupous inflammation. There may be œdema of the glottis.

The Lungs may be congested and œdematous, or hepatized, or the seat of pyæmic infarctions.

The Peritoneum may be inflamed.

The Duodenum may be the seat of perforating ulcers.

The Liver, Spleen, and Kidneys may be the seat of pyæmic infarctions.

DEATH FROM LIGHTNING.

Persons who are struck by lightning may die instantly ; or may continue for several hours comatose, or delirious, and then either die or recover ; or they may die after some time from the effects of the burns and injuries received.

The post-mortem appearances are very variable. Sometimes there are no marks of external violence. Sometimes the clothes are burnt and torn, while the skin beneath them is unchanged. Usually there are marks of contusion and laceration, or ecchymoses, or lacerated, punctured wounds, or fractures of the bones, or superficial or deep burns. The track of the electric fluid may be marked by dark-red streaks on the skin.

The internal viscera may be lacerated and disorganized in the same way.

DEATH FROM POISONING.

THESE examinations should be made with even more than the usual care. The external inspection of the body and the examination of *all* the viscera should be thorough and detailed. Every appearance should be noted at the time and nothing left to the memory.

The stomach and intestines should be removed from the body and opened in separate clean vessels. After examining the appearance of these organs, they should be placed each with its contents in clean glass jars, without the addition of any foreign fluid. Portions of the liver, spleen, kidneys, and brain should also be placed in separate glass jars. All these jars should be closely corked and sealed until they are given to the chemist for his examination.

It is important to remember that many poisons destroy life without producing any lesions, and that many cases of sudden death occur without any discoverable cause and without the possibility of poisoning.

In bodies which are exhumed for examination, the preservation of the viscera for chemical examination is often all that can be done. All the tissues may be so changed that it is impossible to say what lesions have or have not existed.

SULPHURIC ACID.

The effects of this poison vary with the amount taken, and its strength. Death usually takes place in from eighteen to twenty-four hours. Cases are recorded in which death took place in three quarters of an hour. When the poison is less intense, the patient may survive for several months.

The skin of the face about the mouth may be blackened and charred by the acid. The mouth and pharynx are of a grayish or blackish color, while the deeper tissues are reddened. Sometimes these regions escape the action of the poison.

The larynx, trachea, and lungs are sometimes acted on, softened and blackened by the accidental passage of the acid into them. This may even take place when the acid does not pass into the œsophagus.

The œsophagus seldom escapes. It is grayish or blackish colored, softened, and the mucous membrane comes off in shreds. If life is prolonged, cicatrices and strictures are formed.

The stomach is coated on its internal surface with a black, sticky layer beneath which the mucous membrane is reddened. Sometimes perforation takes place, and the acid blackens and softens the adjoining viscera. In protracted cases, cicatrices are formed, and the organ is contracted. If the poison is dilute, there may be only the lesions of chronic gastritis.

The blood is thickened, syrupy, acid, and may form thrombi in the vessels.

The body may be partially preserved from decomposition.

Fatty degeneration of the renal epithelium is mentioned by some authors.

Sulphate of Indigo produces the same lesions as sulphuric acid. It also stains the tissues of a dark blue color.

NITRIC ACID.

The surface of the mucous membrane of the mouth, pharynx, and œsophagus is covered with yellow eschars wherever the acid has touched it. Beneath and around the eschars the tissues are congested and red. The poison may be introduced into the œsophagus without acting on the mouth. The stomach contains a viscous, sanguinolent, yellow or greenish fluid. The mucous membrane is congested, red, swollen, and softened, ecchymotic. It is rarely perforated.

The duodenum may be inflamed, and the inflammation extend to its peritoneal coat. The rest of the intestines usually escapes from the action of the acid.

The larynx is very frequently acted on by the acid. There are yellow eschars, congestion and swelling of the mucous membrane, sometimes cedema of the glottis. The trachea may be inflamed, and the lungs congested.

If the patient survives the first effects of the poison, the lesions of chronic inflammation, cicatrization and contraction are found at a later period.

The acid nitrate of mercury, if taken in a concentrated form into the stomach, produces the same lesions as nitric acid.

HYDRO-CHLORIC ACID.

The lesions are much the same as those produced by nitric and sulphuric acids. The eschars are of a whiter color, and it is more common to find false membranes on the inflamed surfaces.

OXALIC ACID.

The dose of this poison which may destroy life is variable. Sixty grains may prove fatal, an ounce may not cause death.

The same amount of poison does not always cause death at the same period. Three minutes and twenty-three days are the extremes; an hour is the usual time. The mucous membrane of the mouth, pharynx, and œsophagus is white, or coated with brown vomit from the stomach. The stomach contains a dark brown, mucous fluid. The mucous membrane is pale, softened, brittle, and looking as if it had been boiled in water. Sometimes the mucous membrane is red and congested, sometimes it is blackened and gangrenous. If life is prolonged, the whitened condition of the mucous membrane is succeeded by congestion and inflammation.

The duodenum may be inflamed. In some cases of death from this poison, there are no well marked lesions.

Oxalate of potash produces the same lesions as oxalic acid.

TARTARIC ACID.

This acid is seldom used as a poison. In large doses, it may prove fatal.

The lesions in the cases observed were inflammation and redness of the entire alimentary canal.

POTASH.—SODA.

These alkalies and their carbonates are rarely used as poisons. They may destroy life in a few hours, or not until after several weeks.

The mucous membrane of the mouth, pharynx, œsophagus, and stomach is blackened and softened in patches, swollen, congested, and inflamed. The larynx and trachea may also be inflamed and swollen. If life is prolonged, cicatrices and strictures of the œsophagus and stomach are produced.

AMMONIA.

The vapor of strong ammonia may cause death from inflammation of the larynx and air-passages.

The strong solution of ammonia produces corrosion and inflammation of the mouth, œsophagus, and stomach. The larynx, trachea, and bronchi are frequently inflamed, and may be coated with false membranes.

I have seen fatal inflammation of the rectum and colon produced by an enema of strong solution of ammonia.

NITRATE OF POTASH.

This salt may prove poisonous in large doses. In the cases which have been observed, there was intense congestion and inflammation of the stomach, and in one case perforation of that organ.

PHOSPHORUS.

This substance has come into very common use as a poison, especially in Germany. The heads of lucifer matches are the favorite form of administering it. It is more commonly used to effect suicide than to destroy the lives of others.

The poison acts very differently in different cases. In some

cases, the ingestion of the poison is followed in a few minutes by vomiting, and the patient dies in a few hours. In other cases, there is at first vomiting, then very few symptoms until the third day. At that time the vomiting returns, there is jaundice, great prostration, syncope, delirium, and coma. In other cases, again, there may be no symptoms whatever for several days. Not infrequently there are no severe symptoms, and the patient suddenly and unexpectedly dies.

The post-mortem appearances vary according to the length of time which elapses before death.

If death takes place in a few hours, the only lesions are those produced by the direct action of the poison. The mouth, pharynx, and œsophagus usually escape. The stomach may be only slightly reddened, or there may be patches of inflammation and erosion. The contents of the stomach are often mixed with blood, and may have the peculiar smell of phosphorus. There may be little bits of wood present when the poison has been taken on the heads of lucifer matches. It is said that the mucous membrane of the stomach may emit a phosphorescent light in the dark.

If death does not ensue until after several days, the lesions are more marked. The body is usually jaundiced. There may be ecchymoses beneath the pericardium, pleura, and peritoneum, in the lungs, the kidneys, the bladder, the uterus, the muscles, and the subcutaneous connective tissue.

The Stomach usually presents no very striking changes. There may be small circumscribed spots of inflammation and erosion. The most constant change is a granular degeneration of the cells which fill the gastric follicles. In consequence of this, the mucous membrane appears thickened, opaque, of white, gray, or yellow color.

The small intestine appears normal or is congested.

The Liver is found in different degrees of granular and fatty degeneration, and is often stained yellow from the jaundice. It is increased in size and of a grayish, grayish-yellow, or light yellow color, unless stained by the jaundice. Less frequently the centres of the acini are congested, or the entire liver is congested, or there are small hæmorrhages in the liver tissue. The

consistence of the organ is at first flabby; in the later stages, that of the ordinary fatty liver.

The hepatic cells are increased in size, granular, and, after a time, contain small oil-globules. The oil-globules afterward increase in size and number, but do not fuse together to fill each cell with a single oil-globule, as in the ordinary fatty liver. Finally there may be breaking down of the cells into a mass of fat and granules.

In the interstitial tissue of the liver, we may find collections of lymphoid cells, also in the adventitia of the branches of the portal vein, and in the lobules.

The Kidneys.—There is often granular and fatty degeneration of the epithelium of the tubes.

There may be granular degeneration of the voluntary muscular fibres throughout the body and in the heart.

ARSENIC.

From two to three grains of arsenious acid is usually considered a fatal dose. But persons have recovered after taking a much larger quantity.

Death usually takes place in from eighteen hours to three days. But persons have died within twenty minutes, and others not for twenty days.

The mouth, pharynx, and œsophagus may be inflamed, but are more frequently unaltered.

The stomach may be empty, or contain mucus mixed with blood. The arsenic in substance may be found adherent to the mucous membrane. The entire inner surface may be red and inflamed, or there are patches or streaks of congestion. The inflamed patches may be thickened and covered with false membrane. There may be acute gastritis even when the poison is absorbed by the skin, and not introduced into the stomach; Taylor mentions a case in which the coats of the stomach were thickened and gelatinous, but not congested; or there may be degeneration of the gastric follicles as in phosphorus poisoning.

The entire length of the intestine may be congested and inflamed, but usually the action of the poison does not extend beyond the duodenum. In some cases, the solitary and agmin-

ated follicles are swollen, and the intestines contain white, rice-water fluid.

The coats of the stomach are often preserved from decomposition for a long time after death. Death may be produced by arsenic without any well marked lesions.

Arsenite of Copper, Scheele's Green, Paris Green is a favorite article in New-York for producing suicide. It is usually taken in considerable quantities, and is often found in the stomach after death. The lesions are the same as those produced by arsenious acid.

The sulphide and chloride of arsenic are sometimes used as poisons. The lesions are the same as those of arsenious acid.

CORROSIVE SUBLIMATE.

The mucous membrane of the mouth is swollen and whitened.

The œsophagus is swollen and white, or congested, or is unaltered.

The stomach is usually contracted. There are inflamed and congested, sometimes gangrenous, patches of the mucous coat. Perforation is said to have occurred in one case. Small ecchymoses in the mucous coat are not uncommon. Sometimes hardly any change can be appreciated.

The intestines may appear normal, or there may be patches of congestion and ecchymoses. The larynx and trachea may be red and congested.

The kidneys may exhibit granular and fatty degeneration of the epithelium.

LEAD.

The different preparations of lead may prove fatal either from the immediate effect of large doses, or from the gradual effects of repeated small doses. Although there may be marked symptoms during life, the post-mortem lesions are few and variable.

Large doses may produce acute gastritis. The kidneys are sometimes the seat of fatty degeneration. Chronic lead poisoning may lead to chronic meningitis and changes in the brain substance.

COPPER.

Sulphate of copper and verdigris are sometimes employed as poisons. The post-mortem appearances have only been observed in a moderate number of cases. The pharynx and œsophagus may be unchanged, or be somewhat inflamed. The stomach may be unchanged, or bits of the copper may adhere to its mucous coat; and there may be patches of inflammation, of gangrene, and even of perforation.

TARTAR EMETIC.

This preparation of antimony may prove fatal when administered in a single large dose, or in repeated small doses.

The post-mortem lesions are not constant. In cases of chronic poisoning there are usually no appreciable lesions.

In cases of acute poisoning there may be evidences of acute inflammation of the œsophagus, stomach, intestines, and peritoneum. The larynx may be congested, and the lungs may be engorged with blood. But none of these lesions are constant.

VEGETABLE IRRITANTS.

Aloes, colocynth, gamboge, jalap, scammony, savin, croton-oil, colchicum, veratria, turpentine.

All these drugs sometimes produce poisonous effects. Vomiting, purging, and prostration are the usual symptoms. Congestion and inflammation of the stomach and intestines are the lesions which would be naturally looked for; but these lesions are sometimes present, sometimes absent.

CANTHARIDES.

This substance may be given in powder or in tincture.

The entire length of the alimentary canal may be found congested and inflamed. The kidneys, ureters, and bladder may be congested and inflamed.

OPIUM.

The post-mortem appearances in persons who have been killed by any of the preparations of opium are negative. Congestion of the brain and of the lungs are spoken of by most authors, but they seem to depend principally on the way in which the patient dies, rather than on any specific action of the drug.

HYDROCYANIC ACID.

Fatal doses of this poison destroy life in a very few minutes.

The skin is usually livid, the muscles contracted. The stomach may be congested, and the venous system generally is unusually full of blood. The most characteristic condition, when it is present, is the odor of the acid exhaled from the stomach and tissues. This odor may remain for a long time after death. Cyanide of potash destroys life in the same way as hydrocyanic acid. The lesions are equally uncertain. The tissues and stomach often exhale the same smell of bitter almonds.

Nitro-benzole produces the same symptoms and lesions as prussic acid. The odor of bitter almonds in the tissues is even more intense.

ALCOHOL.

The different preparations of alcohol, when taken in large quantities, sometimes produce sudden coma and death in from half an hour to several hours.

The bodies are said to resist decomposition for an unusual length of time. The stomach and tissues often have an alcoholic smell. There is congestion, and sometimes extravasation of blood in the brain and its membranes. The veins everywhere are full of blood. The bladder may be distended with urine.

Chronic alcoholic poisoning is of a different nature. The subjects of it may die from some other disease, or they die after a debauch without any thing else to account for their death. In the latter case, there may be *delirium tremens*, or the patient dies exhausted and comatose. Chronic alcoholism is not infre-

quently mistaken for meningitis. The dura mater is frequently adherent to the skull. The pia mater is thickened and white, sometimes œdematous. The brain usually appears normal.

The lungs are usually congested.

The heart is thickly covered with fat, and flabby. The stomach presents the lesions of chronic gastritis.

The liver is fatty, cirrhotic, or both.

The kidneys are in a condition of simple granular or fatty degeneration, or the seat of diffuse nephritis.

CHLOROFORM.—ETHER.

Chloroform may cause death when it is inhaled, and when it is taken in a fluid form into the stomach.

Death from the inhalation of chloroform has become a tolerably frequent accident in surgical practice. A very considerable number of post-mortem examinations of such cases have been recorded. They show that there is no constant or characteristic lesion, nor is there any satisfactory explanation of the way in which such persons meet their death.

Death from the swallowing of liquid chloroform is rare. Taylor gives a case in which a drachm killed a boy four years old. Adults can take with impunity a much larger quantity. I have seen one case in which four ounces proved fatal to a woman in a few hours. After death, there was a strong odor of chloroform when the stomach and duodenum were opened. The stomach was half full of undigested food. There were no lesions. Other cases are reported in which there was inflammation of the stomach, and one case in which the liquid had passed into the larynx and trachea, producing intense inflammation.

Ether, given by inhalation, occasionally causes death. The post-mortem signs are negative.

There is one death recorded by Casper, from its use in a fluid form. The cavities of the body exhaled a strong smell of ether; otherwise, there were no lesions.

STRYCHNIA.—NUX VOMICA.

We possess very few good records of post-mortem examinations after death from these poisons. The contraction of the muscles, which exists during life, disappears after death. There may be excessive congestion of the membranes of the brain. Casper mentions a peculiar violet color of the muscles of the pharynx and œsophagus. Otherwise there are no lesions.

CONIUM, ACONITE, BELLADONNA, LOBELIA INFLATA, DIGITALIS, STRAMONIUM, VERATRUM.

These vegetable poisons are administered in their natural form of leaves, berries, and roots, or in tinctures, infusions, and extracts, or in the form of their active alkaloid principles.

If the leaves, berries, or seeds are given, they may be detected in the contents of the stomach by microscopic examination. Otherwise the results of autopsies are unreliable.

The brain and its membranes, and the lungs, may be congested. The stomach may present patches of congestion, inflammation, and extravasation, or its entire mucous coat may be inflamed, or it may appear normal.

CARBONIC OXIDE.

This gas is produced from burning charcoal, and forms the poisonous ingredient of illuminating gas. It is usually used as a poison in one of these forms.

The most characteristic post-mortem appearance is that of the blood, which is of a brilliant red color. The muscles and viscera also appear red from the color of the blood in them.

The viscera present the same changes as in death from suffocation.

CARBOLIC ACID.

A number of cases of death from this poison have been reported during the last few years. It acts as a corrosive poison on the mucous membrane of the mouth, pharynx, œsophagus, stomach, and intestines, producing white eschars with intense congestion. The lungs are usually intensely congested.

PART IV.

TUMORS.

TUMORS.

ANY classification of tumors which can be given, at the present time, is in great measure a provisional one. Our knowledge of these growths is not yet sufficient to enable us to speak of them with certainty. There can be no doubt, however, of the proper method in which the study of tumors should be pursued in order to arrive at more complete results. We know that all new growths are composed of the same anatomical elements as exist in the normal tissues, although their arrangement and mode of growth may be different. We know that no anatomical element, whether existing in a normal tissue or in a pathological new growth, has any inherent malignant or benignant properties. We know that no grouping or arrangement of such indifferent elements can give them any specific properties. The only scientific plan, therefore, is to begin at the beginning; to find out, by patient study, what is the exact structure, the mode of growth, and the relationship to the surrounding tissues of every new growth which can be observed. To this we must add in each case the clinical history, the age and condition of the patient, the situation of the new growth, whether it recurs after extirpation, whether it grows slowly or rapidly, whether it invades neighboring tissues, whether metastatic tumors of the same nature are formed in other organs. By an accumulation of such facts as these, we arrive nearer and nearer to an exact knowledge of tumors. The number of existing observations is already sufficient to enable us, from the anatomy of a tumor, its situation, and the history of its growth, to give a prognosis with greater certainty than can be done in most diseases.

To classify tumors, therefore, we merely group them according to our present knowledge of their anatomy, with the expectation that this grouping is to be much changed by future investigations. We are especially hindered at the present time:

by the prevailing uncertainty as to the exact anatomy of connective tissue.

The classification and names of tumors which I give are, with some modifications, those proposed by Virchow, and adopted at the present day by most pathologists.

We divide tumors into four classes :

- I. The Cysts.
- II. The simple Tissue Tumors.
- III. The Composite Tissue Tumors.
- IV. The Cellular Tumors.

I. THE CYSTS.

Cysts are of three kinds : (1.) Those which are formed in pre-existing cavities. (2.) Those which are new formations. (3.) Those which are formed by an abnormal development of the foetal tissues.

(1.) Those which are formed in preëxisting cavities : Extravasation, exudation, and retention cysts.

The extravasation cysts are formed by the escape of blood into the serous sacs and natural cavities of the body. They are also called hæmatoma and hæmatocele.

The exudation cysts are formed by the exudation of serum into the serous sacs and natural cavities of the body. They are also called hydrocele and hygroma.

The retention cysts are formed by the retention of the excretions of glands and the consequent dilatation of their follicles and ducts. They are subdivided into follicular cysts, mucous cysts, and retention cysts of large ducts.

To the follicular cysts belong the forms called comedo, milium, molluscum, and atheroma. Atheroma (encysted tumor) is the only one of these which is usually classed with the tumors. It is formed by the closure of the mouth of a hair follicle. In consequence of this, the hair follicle itself, and the sebaceous glands which open into it, are dilated. In this way is formed a cyst with a smooth wall and cheesy contents. The contents are made up of fat, epidermis, granular matter, and cholesterine.

The Mucous Cysts are formed by the dilatation of mucous glands. They may be single, or composite from the dilatation of several follicles. They may project so as to form a sort of polypus. They are found in the mucous membrane of the mouth, pharynx, nose, antrum, cervix uteri, vagina, and alimentary canal.

The retention cysts of larger canals are formed by dilatation of the ducts of the salivary glands, the pancreas, the mamma, the tubes of the kidney, etc.

(2.) The cysts of new formation.

(a.) Cysts produced by softening.

Portions of solid tissue, either normal or pathological, may become softened and fluid, and in this way cysts be formed. Thus we find cysts full of fluid fat in fatty tumors; cysts full of mucus or serum in fibromata, chondromata, and myomata. In the sarcomata and carcinomata, this process is especially common. The most common ways in which the process takes place are by mucous degeneration of the tissue, or by a sort of circumscribed œdema.

With these cysts may also be classed those formed in the thyroid gland, and in adenomata of the ovaries by a colloid degeneration of the cells lining the gland follicles.

(b.) Cysts, properly speaking, cystoma.

These cysts may be produced by the irritation of extravasated blood, or of a foreign body.

Or continued pressure on some part induces irritation and an accumulation of serum. Thus are formed the false bursæ under corns, etc.

Or cysts are formed as new growths in connective tissue in methods which are yet unknown to us. It is probable that some cysts are formed by dilatation of the lymphatic vessels.

(3.) *Dermoid Cysts*.—In these cysts, the internal surface of the cyst-wall has the same structure as the skin. We find in the cyst-wall hair and sebaceous follicles, sweat-glands, papillæ, and an internal covering of epithelium. The cysts contain a grumous fluid in which are epithelium, fat, cholesterine, hair, teeth, cartilage, and bone.

It is now generally held that these cysts are formed before

birth by some change in the development of the tissues of the fœtus. They are usually situated in the subcutaneous tissue. They are found in the orbit, in the floor of the mouth, in the neck, and less frequently in other regions. They are also found in the ovaries, the testicles, the membranes and the substance of the brain, the eye, the anterior mediastinum, the lungs, the mesentery and omentum, and around the uterus.

Cysts lined with ciliated epithelium are found in the brain, the external ear, the liver, and the testicles. They have probably the same nature as the dermoid cysts.

II. SIMPLE TISSUE TUMORS.

Fibroma,
Myxoma,
Lipoma,
Chondroma,
Osteoid-Chondroma,
Osteoma,
Myoma,
Glioma,
Psammoma,
Neuroma.

I. *Fibroma*.—Fibrous tumors.

These are tumors composed of connective tissue fibres and cells; the cells in small numbers. According to their external form we distinguish:

(1.) *Fibroma nodosum*.—The tumors are rounded and nodular, composed of dense fibrous tissue.

(2.) *Fibroma diffusum*, or Elephantiasis.—There is a diffused new growth of fibrous tissue from the deep layers of the cutis.

(3.) *Fibroma papillare*, Papilloma.—The tumors are hypertrophies of normal papillæ, or grow from surfaces where no papillæ exist. They form papillary, or polypoid growths, often composed of branching tufts. They consist of a central stroma of connective tissue, often of the branched variety, with many round cells, and are covered with epithelium. These tumors grow

from the surface of the skin and of the mucous membranes, from the internal surface of the milk-ducts in the mamma, and from the walls of some cysts. They constitute a large part of the growths commonly called polypi.

All the fibromata are, as a rule, local growths of a benignant character.

II. *Myxoma*.—Fibro-cellular tumors, colloid.

This name is given to tumors which are formed of mucous tissue. Mucous tissue consists of round, fusiform, and stellate cells imbedded in a gelatinous, transparent, mucine-yielding basement substance. It is found in the umbilical cord, in the foetus as the first stage of adipose tissue, and in the adult in the vitreous body.

The myxomata are tumors of irregular form, lobulated, having a peculiar soft, gelatinous, semi-transparent appearance. They are identical in their minute structure with that of normal mucous tissue. They are but seldom found as pure myxomata. It is much more common to find combinations with lipoma, chondroma, fibroma, and sarcoma.

These tumors occur most frequently in adult life, but are sometimes congenital. They usually grow as isolated tumors, except on the nerves, where they are multiple. They grow slowly; may attain a large size; are either inclosed in a fibrous capsule, or invade the neighboring soft parts.

The pure, uncombined myxomata have a merely local significance, but sometimes recur after extirpation. The combination forms with sarcoma, etc., have, on the other hand, well marked malignant characteristics, recur after extirpation, and form metastatic tumors.

These tumors occur most frequently in the adipose tissue of the thigh, cheek, nates, hand, labia, mesentery, peritoneum, and pelvis of the kidney. In the female mamma, as intra-canalicular growths; in the parotid gland; in the brain, spinal cord, and nerves; and in the bones, especially the maxillary bones, these tumors are not infrequently observed. The degeneration of the chorion, commonly called the hydatid mole, is classed by Virchow with these tumors. The umbilicus in children sometimes gives rise to these tumors.

III. *Lipoma*.—Fatty tumors.

This name is given to tumors composed of adipose tissue ; that is, of a stroma of connective tissue containing fat-cells.

These tumors are of irregular shape, always lobulated, soft, movable, and may attain a great size.

They may undergo calcification, ossification, may soften so as to form oil-cysts, and may become the seat of chronic inflammation and suppuration.

The most important combination of the lipomata is with the myxomata.

The lipomata may be single or multiple. They occur most frequently in adults, but are found in all ages and congenitally. They are sometimes hereditary. They are purely local, benign tumors.

They arise in preëxisting adipose tissue. They are found very frequently in the subcutaneous adipose tissue in all parts of the body ; between the muscles ; beneath the serous membranes, especially the peritoneum ; beneath the synovial membranes, protruding into the cavities of the joints ; beneath the mucous membrane of the mouth, trachea, bronchi, stomach, intestines ; in the female mamma ; in the kidneys ; in the pia mater ; and in the scrotum and labia. These tumors, when growing beneath the skin, or the synovial, mucous, and serous membranes, may assume a polypoid form with a narrow pedicle. This pedicle may sometimes atrophy and leave the tumors free in the peritoneal or joint cavities.

IV. *Chondroma*.—Cartilaginous tumors.

These tumors are composed of cartilage. The cartilage-cells may resemble those of normal cartilage, or may be deformed. The basement substance is hyaline, fibrous, mucous, or calcified. The tumors are inclosed in a fibrous capsule, lobulated, and divided up by bands of fibrous tissue.

We may distinguish two varieties :

The Ecchondroses and the Enchondromata. The ecchondroses grow from the permanent cartilages of the ribs, the synchondroses, the inter-vertebral cartilages, and the cartilages of the larynx and trachea. They only reach a small size, and have but little importance.

The enchondromata most frequently grow from the medulla, or periosteum of the bones; in the salivary glands, the testis and mamma, but are also found in other regions. They may be single or multiple. They may reach a very large size. They are usually local growths of a benign character; but cases of recurrence and of metastatic tumors have been observed.

V. *Osteoid-Chondroma*, Osteoid Cancer.—These tumors are composed of bone cartilage, such as we see normally existing as a stage of the ossification of the flat bones of the cranium. The cells are smaller than cartilage-cells, the intercellular substance is dense, or calcified. They are found especially at the ends of the long bones. Local recurrences after extirpation and metastatic tumors are not uncommon.

VI. *Osteoma*.—The formation of new bone, and the increase in size of normal bones, are of common occurrence. Only those bony growths, however, are classed among the tumors, which have a considerable size, are distinctly circumscribed, and are not the result of inflammatory processes. The tissue of these tumors is the same as that of normal bone. The bone may be compact and hard, or cancellous and spongy, or separated by cavities containing medulla.

Many of the fibrous and cartilaginous tumors may become ossified partially or entirely. Combinations of fibroma, chondroma, sarcoma, and carcinoma, with osteoma, are not uncommon.

The osteomata occur either singly or may be developed in large numbers at once. They are found at all periods of life, and are sometimes congenital. They grow slowly and interruptedly, usually without pain. They may be hereditary, or be caused by a blow, or may grow at the seat of a fracture.

Eberth describes the case of a boy, in whom sixty-five osteomata were developed in the course of a year. The tumors appeared in groups, and each group was attended by a severe attack of fever.

The osteomata are tumors of purely local significance and benign character.

They grow from the bones, or from the soft tissue. Those

which grow from the bones are usually called exostoses, or, if they arise from the medullary cavity, enostoses.

The exostoses are developed from the periosteum, and may occur on any of the bones.

On the long bones, near the insertions of the tendons, exostoses sometimes occur, which resemble the epiphyses. These exostoses grow out in a lateral direction, are formed of cancellous bone containing medulla, are covered by a thin layer of cartilage, and may even be surrounded by a synovial sac. The name of *Exostosis cartilaginea* has been given to them.

In the soft parts, osteomata are less frequent. They are found near to, but not connected with the bones. As small tumors of little significance, they are met with in the connective tissue in different parts of the body.

VII. Myoma.—This name is given to those tumors which are composed principally of muscular tissue.

Such tumors may be composed either of voluntary or of organic muscular fibres. The two varieties are called respectively *Myoma striocellulare*, and *Myoma lævicellulare*.

Myoma Striocellulare.—These tumors are of very rare occurrence. They have only been observed in the heart. Here they occur in the ventricular walls as single or multiple tumors.

Such tumors have been observed three times in new-born children, and once in an adult. A new growth of striated muscle in composite tumors, especially in the testicle, is more frequent.

Myoma Lævicellulare.—This form of myoma is, on the other hand, very common. The tissue resembles that of organic muscle, long fusiform-cells running in parallel bundles, and mixed with a variable amount of connective tissue. The muscular cells may be very abundant, and the tumor be soft, of a loose, areolar texture and pinkish color; or the fibrous element may be in excess, and the tumor be of a stony hardness, compact and white.

These tumors may undergo calcification; portions may soften and break down so as to form cystoid cavities; serum may accumulate in the soft, areolar varieties so as to form cysts; portions may become gangrenous; fatty degeneration may occur;

the muscular elements may atrophy and disappear, leaving only fibrous tissue; or the blood-vessels may become dilated, and form a cavernous tumor.

Combinations of myoma lævicellulare, with cysts, sarcoma, and carcinoma, are not infrequent.

The myomata may be single or multiple. They occur most frequently after middle age. Their growth is slow, and they may become stationary, or even atrophy. Their significance is purely local.

The most frequent seat of these tumors is the uterus. Here they are found usually at the fundus, are multiple, and are intra-parietal, submucous or subserous. The subserous and submucous varieties often have a polypoid form, with a small pedicle. This pedicle may atrophy, and the tumor be left unconnected with the uterus. In the cervix uteri, vagina, broad ligaments and ovaries, these tumors are less common.

Less frequently, myomata are found in the œsophagus, stomach, and intestines. A certain number of the enlargements of the prostate are due to the growth of myomata in this organ.

VIII. Glioma.—This name is given to tumors which are composed of neuroglia. The neuroglia is the connective tissue of the brain. It is composed of a basement substance, which is amorphous and finely granular, or fibrillated, and of small, pale, round and oval cells, imbedded in the basement substance.

The gliomata are composed of this same tissue, but with an increased number of cells. They are found in the brain, cranial nerves, and spinal cord. They look rather like hypertrophies of some portion of the brain than like separate new growths. Their consistence is soft and mottled by small hæmorrhages, or hard, with a denser basement substance.

There is a form of retinal tumor, to which the name of glioma is also usually given, but which seems to belong more properly to the sarcomata.

IX. Psammoma.—This name has been given to a form of tumor which occurs in the brain, usually growing from the dura mater. The growths are composed of bands of fibrous tissue, of small, round cells, fusiform cells, and cells

resembling the epithelium of the dura mater; of blood-vessels, which may be solid or calcified; and of peculiar rounded, laminated bodies, which are partly or entirely calcified. These latter bodies are found normally in the ependyma and choroid plexus of the brain, where they are called brain-sand. It is from them that the tumor is named. Although these tumors are of not very infrequent occurrence, and many observations of them have been published, their real nature is still very uncertain. It is convenient, for the present, to give them all this common name, although it is quite possible they may belong to different classes.

X. Neuroma.—This name is applied to tumors which are composed of nervous tissue. Such tumors are found on the course of the nerves. They are not to be confounded with tumors of different structure—fibromata, myxomata, etc., which also occur on the nerves.

We distinguish two varieties according to the character of the nerve fibres. Neuroma myelinicum, composed of nerve-fibres, in which the white substance of Schwann is present.

Neuroma amyelinicum, composed of small, gray nerve fibres, without the white substance of Schwann.

Those of the first kind are much the easiest to recognize and demonstrate.

Those of the second kind resemble very closely connective tissue.

III. COMPOSITE TISSUE TUMORS.

These are tumors composed, not merely of a simple tissue held together by connective tissue, but of several different tissues forming an organoid growth. We distinguish :

Angioma,
Adenoma.

I. Angioma.—This name is given to tumors which are principally composed of a new growth of vessels. We distinguish three varieties : The Simple Angioma, the Cavernous Angioma, and the Cavernous Lymph-Angioma.

(1.) The Simple Angioma. These tumors are formed of a new growth of capillary vessels. They are situated in the skin, or in the subcutaneous tissue. The angiomata of the skin (nævi) form flattened, red growths in the skin, not separated from the surrounding tissue, and gradually replacing the skin tissue. Those in the subcutaneous tissue are more circumscribed and nodular. They may be joined with a new growth of fatty tissue. The simple angiomata are, as a rule, congenital.

(2.) The Cavernous Angioma is formed of a connective tissue stroma in which are numerous cavities, communicating with each other, and filled with blood. The inner surface of these cavities is lined with endothelium. The blood in the cavities is usually dark-colored and venous. They communicate with small arteries and veins, or with large arteries and veins; they may pulsate. They are inclosed in a fibrous capsule, or are continuous with the surrounding tissues.

These growths are congenital, or are formed at any period of life.

(3.) Lymph-Angioma.

A circumscribed new growth of lymphatic vessels sometimes occurs. The tumor has a cavernous structure, with its cavities filled with lymph, instead of blood. It forms part or the whole of some of the hypertrophies of the tongue, lips, and cheeks. It is usually a congenital condition.

II. *Adenoma*.—This name is given to tumors which are composed of glandular tissue, not including the lymphatic glands. This is a group of tumors of which the limits and character are not yet well determined. They are closely related in structure to some of the forms of epithelial cancer, and yet many of the growths seem to be entirely benign.

The new glandular tissue follows the type of that of the region where it is situated. Sometimes it is a simple hypertrophy of the normal glands of the part.

In the skin, we find adenomata formed of the sebaceous glands and the sweat-glands. In the mucous membranes of the nose, stomach, intestines, rectum, gall-bladder, and cervix uteri, we find new growths of glandular follicles lined with cylindrical epithelium.

In the mamma, there are new growths of mammary glandular tissue.

In the ovaries, most of the compound cysts are adenomata with dilatation of the follicles. The ordinary bronchocele is an adenoma of the thyroid gland.

Combinations of adenoma with sarcoma and papilloma are not infrequent.

IV. CELLULAR TUMORS.

This general and indefinite name is used, because it is general and indefinite, to group together a number of tumors of which we cannot yet decide the exact relationship. We include under it the following varieties :

Tubercle,
Syphilitic Gummata,
Lupus,
Lymphoma,
Sarcoma,
Carcinoma.

I. *Tubercle*.—This name is now generally applied only to the small, gray, miliary granulations, which we find in the serous and mucous membranes and in many of the viscera. Even of these bodies we find different descriptions by different authors.

In the pia mater, the little tumors are formed by an accumulation of lymphoid cells between the perivascular sheath of the small vessels and their outer coats.

In other situations, the growths are made up of lymphoid cells, fusiform cells, large, many-nucleated bodies, (myéloplaxes,) large flat cells with a single nucleus, and a delicate reticulum of branched fibrous tissue.

Cheesy degeneration begins early at the centre of each little nodule.

II. *Syphilitic Gummata*.—These growths stand on the border line between inflammatory growths and new growths proper.

They form tumors from the size of a pin's head to that of a man's fist. They are composed of small, round, oval, and fusi-

form cells, which have a strong tendency to undergo cheesy and fibrous degeneration. The larger tumors are formed by an aggregation of smaller growths. They are frequently associated with a new growth of fibrous tissue.

They have been observed in nearly all parts of the body.

III. *Lupus*.—This new growth is usually treated of with diseases of the skin.

It consists in the formation of small round cells in the deep layers of the cutis. The cell-growth goes on until all the tissues of the skin are replaced by the round cells; then the epidermis falls off, and ulcers are formed. These ulcers may become deep and broad, and if they cicatrize and contract, produce great deformities.

The gross appearances vary according to the size and distribution of the nodules of new growth, and their tendency to ulceration.

IV. *Lymphoma*.—This name is given to tumors which are composed of tissue like that of the lymphatic glands, that is, of a delicate reticulum of branched fibrous tissue holding together a great number of small, round cells.

The growth occurs as an hypertrophy of normal glands, and as an heteroplastic growth in connective tissue and in the viscera.

V. *Sarcoma*.—This name is given to tumors which are principally composed of connective-tissue cells. According to the different forms of connective tissue, we can distinguish Sarcoma proper, composed of round, oval, fusiform, and stellate cells, with a fibrous basement substance.

Myxo-Sarcoma, composed of the same cells, the stellate form predominating, and of a mucous basement substance.

Chondro-Sarcoma, composed of cartilaginous tissue, but with the cells greatly in excess.

Osteo-Sarcoma, composed of osteoid tissue, with the cells in excess.

Glio-Sarcoma, composed of neuroglia, with the cells in excess.

As it is customary to speak of connective tissue proper and of the connective-tissue group of bone, cartilage, mucous tissue, and neuroglia, so we may also speak of sarcoma proper,

having its type in connective tissue proper, and of myxo, chondro, osteo, and glio-sarcoma as belonging to the same group.

Of sarcoma proper we also distinguish several varieties, according to the character of the cells and the amount of the basement substance. Thus, a sarcoma entirely composed of cells, with a very scanty stroma, is called a "medullary sarcoma." If the cells are nearly all of the spindle-shape, a sarcoma-fusocellulare. If there are many of the large nucleated bodies called myélopaxes, a sarcoma gigantocellulare, etc. If the stroma is abundant, and the cells less numerous, we call it a fibro-sarcoma.

Besides these forms, we also find tumors in which there are other anatomical elements in addition to those belonging to the connective tissue. A tumor may be partly formed of muscular tissue, myo-sarcoma; of glandular tissue, adeno-sarcoma; of fatty tissue, sarcoma lipomatodes, etc.

Still further, Virchow has given the name of lympho-sarcoma to a group of tumors which begin in the lymphatic glands and have the same structure as them, but have the clinical history of sarcoma.

These tumors may grow from any point of the body where connective tissue exists. They are of very frequent occurrence. The group is a large one; and while there are some general laws which all obey, there are many modifications.

Generally speaking, these tumors are, when of small size and of short duration, merely local growths. If completely removed, they may never recur, or not until after several years. When they grow quickly, become of large size, and invade the neighboring tissues, they are very malignant; recur soon after extirpation, and form metastatic tumors in the viscera.

The prognosis varies also with the situation of the tumor and with the age and condition of the patient.

In the case of any given tumor, therefore, in order to give an intelligent prognosis, it is necessary to know the situation of the tumor, the time which it has been growing, and the age of the patient, as well as the anatomical characters of the growth.

The different forms of sarcoma have been described under different names by various authors. Fibro-plastic tumor,

myeloid, tumeur à myéloplaxes, encephaloid, cancer, etc., are some of the names which have been employed.

VI. *Carcinoma*.—This class of tumors is one of the most important and, at the same time, one of the least understood. The name was formerly used to designate any malignant tumor; now most authors endeavor to limit it to a particular form of growth.

Generally speaking, we may say that these growths are composed of a connective tissue stroma containing cavities filled with cells, which resemble epithelial cells. Some authors lay the most stress on the arrangement of the cells; others on their shape. Some hold that the new growth arises from connective tissue; others that it arises from normal epithelium. The prevailing idea just at present is that, both in the normal growth of the body and in the formation of pathological new growths, the different tissues are developed from the different germinal layers, and do not intermix. Consequently new growths of epithelium can only arise from preëxisting epithelium.

We may distinguish three varieties: Epithelioma, true carcinoma, colloid carcinoma.

(1.) Epithelioma. Although at present the tendency is to call all the carcinomata epithelial growths, it is convenient to group together a certain number in which this structure is most regular. The epithelial cancers follow the type of the epithelium near which they grow. Those of the skin, mouth, and vagina are formed of large pavement epithelium. Those of the nares, larynx, alimentary canal, and bladder are formed of cylindrical epithelium. Those of the conjunctiva are formed of small pavement epithelium.

The large pavement epithelial cells are usually packed closely together in large, irregular follicles, some of the cells being packed into concentric nests.

The cylindrical epithelial cells are more regularly disposed in tubular follicles, or covering tufts.

The small pavement epithelium usually covers small tufts.

These epithelial cancers resemble the adenomata on the one side, and the true carcinomata on the other. They are usually

looked upon as less malignant than the true carcinomata, but their prognosis is by no means good.

(2.) True carcinoma consists of a connective tissue stroma and cells. If the stroma is abundant and dense, the growth is called scirrhus; if the cells are abundant and the stroma scanty, the growth is called medullary. The cells do not exactly resemble normal epithelium, although they have that general character, but are often malformed in various ways. They are contained in regular tubular, ovoid, or rounded alveoli; or, if the stroma is scanty, appear irregularly distributed.

The prognosis of these growths is bad.

(3.) Colloid Carcinoma. These growths have a gelatinous appearance and consistence. They are formed of a fibrous stroma, which forms small, rounded cavities filled with colloid matter and cells. The cells resemble pavement epithelium; they are numerous, few, or absent. The colloid matter is said to be formed by a change in the cells; but in some of the smallest and youngest growths, we find alveoli filled with colloid matter and no cells.

The prognosis of these growths is bad.

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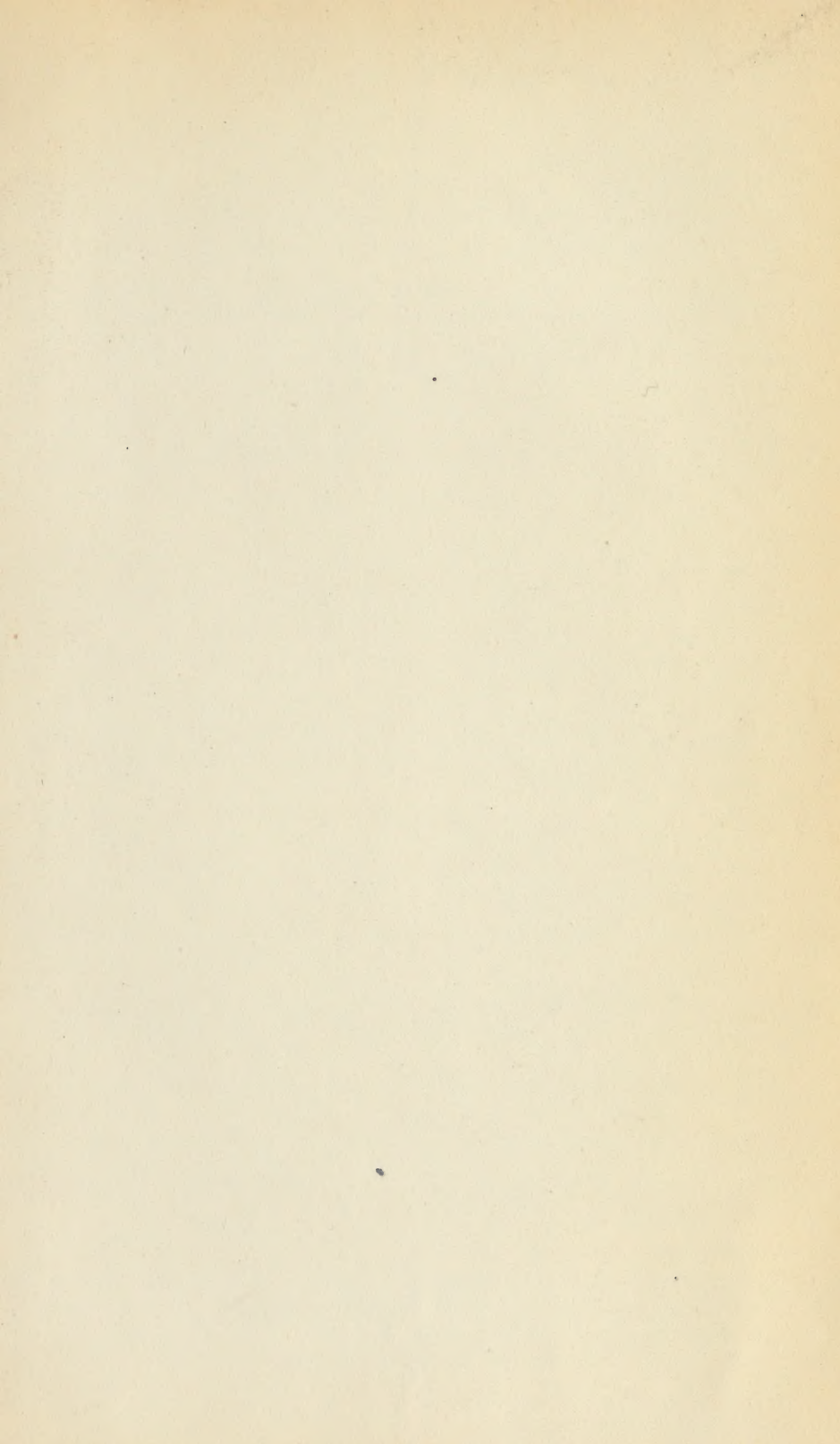
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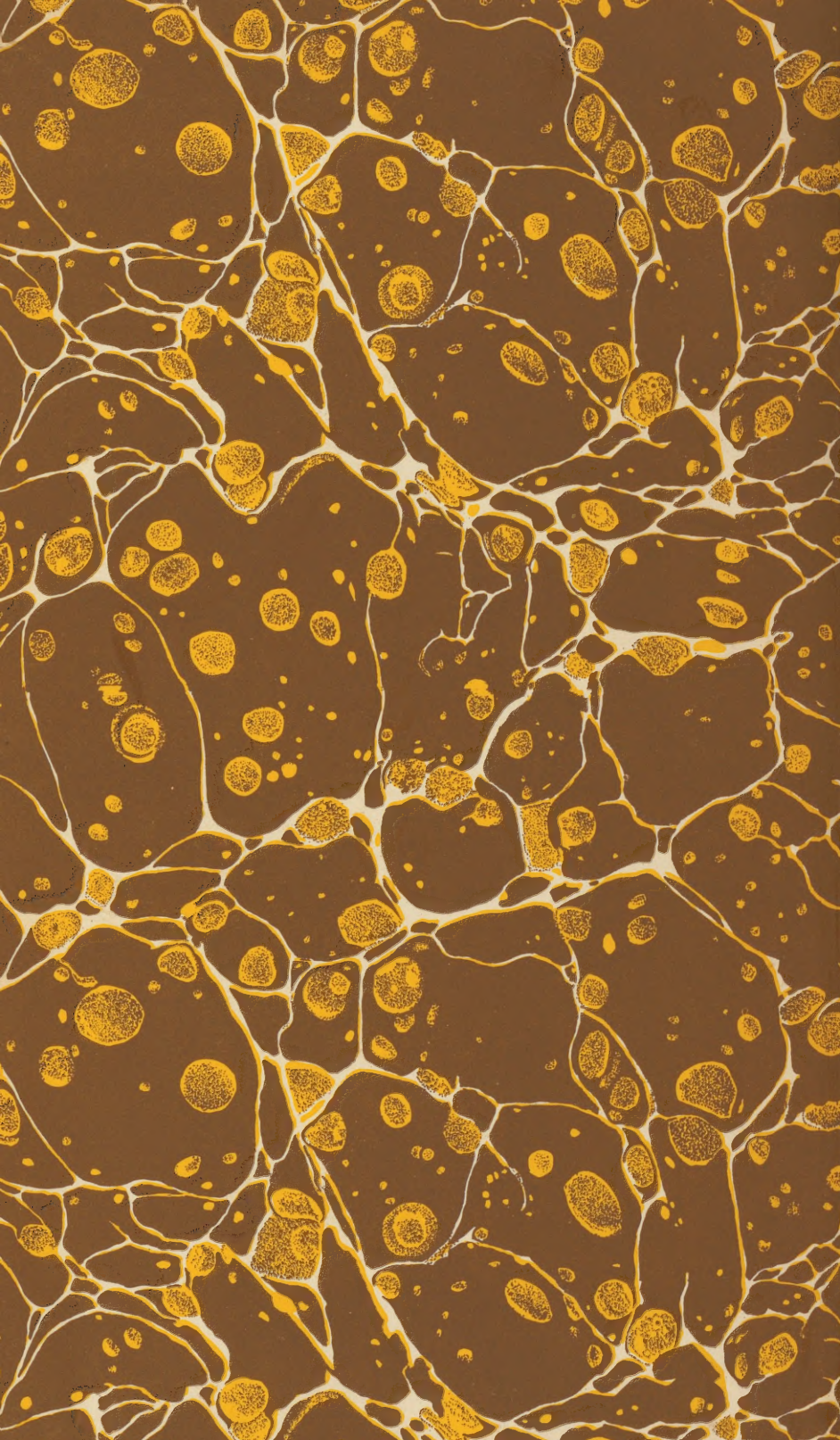
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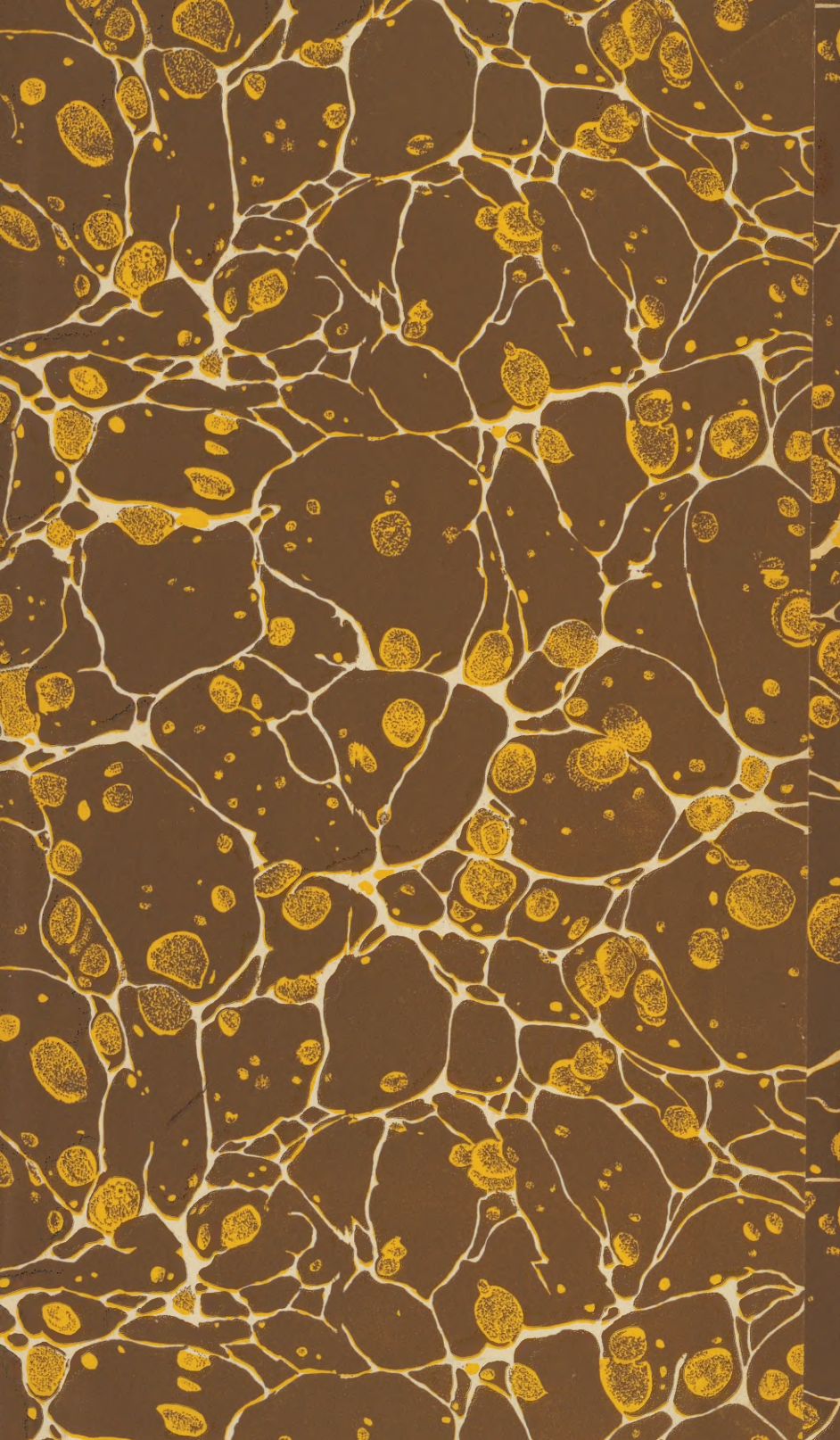
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